BRAIN.

PART IV., 1901.

Original Articles and Clinical Cases.

A STUDY OF THE DEGENERATIONS OBSERVED IN THE CENTRAL NERVOUS SYSTEM IN A CASE OF FRACTURE DISLOCATION OF THE SPINE.

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AND

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With Photomicrographs by E. J. Worrall, M.D.

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For the opportunity of recording the following case we are greatly indebted to the kindness of Dr. Cecil Lyster, under whose care the patient was treated at the Bolingbroke Hospital. As it was clear that the accident must have caused a complete destruction of the spinal cord at and below the level of the third lumbar segment, and as the patient survived for a period just within six weeks, it seemed certain that by application of the Marchi method it would be possible to trace completely, besides the posterior column fibres, the following tracts :—

(1) The direct cerebellar tract of Flechsig or the *fasciculus spino-cerebellaris dorso-lateralis*. (Barker.)

(2) Gowers's tract or the *fasciculus spino-cerebellaris ventralis.*

(3) The *fasciculus spino-quadrigeminalis.*[1] } Mott. Patrick.
(4) The *fasciculus spino-thalamicus.* } Tooth.

The following is a brief clinical abstract of the principal clinical facts of the case :—

Case of W. L., aged 21, *Bolingbroke Hospital.*

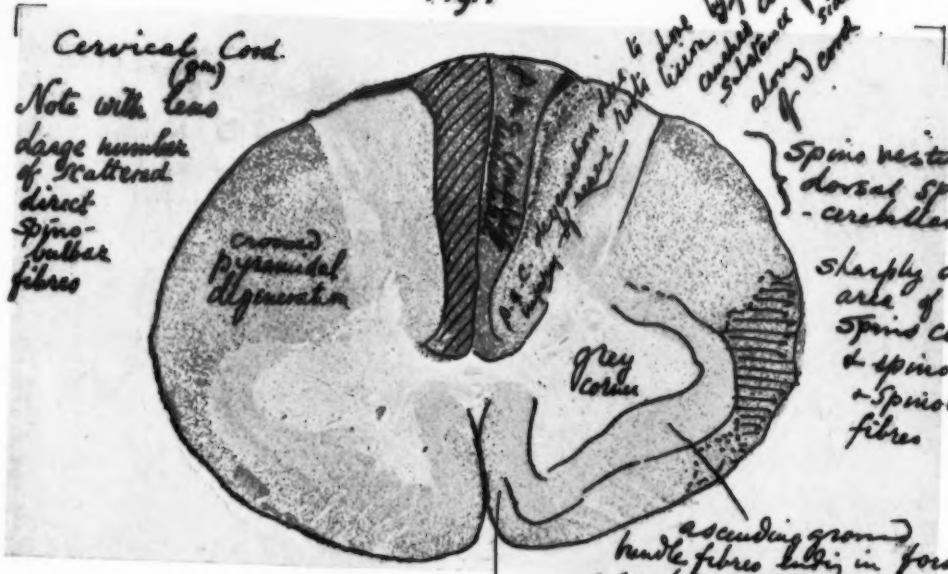
The patient was brought to hospital having been knocked down by an engine and crushed against the edge of the platform. On admission, besides (1) a severe scalp wound in occipital region, and (2) dislocation of the right astragalo-scaphoid articulation with a contused wound of left calf, there was (3) fracture dislocation of the spine in the lower dorsal region. There resulted immediate and complete paralysis and anæsthesia of both legs; all reflexes were absent, and the anæsthesia extended up to the umbilicus. Retention of fæces and urine existed from admission. Cystitis supervened, and though the wounds in the leg were actively treated they showed no tendency to heal. The wasting of both legs and thighs became extreme. No reflexes were ever obtained. The patient died forty days after the accident.

The brain and spinal cord were removed by Mr. H. J. Curtis, F.R.C.S., under difficult circumstances, and for whose assistance in this particular we are indebted. The central nervous system was fixed in formalin 5 per cent. solution and Müller's fluid in equal parts, and subsequently in Müller's fluid alone. For the osmic stain Busch's fluid was employed (solution of osmic acid in iodate of soda solution).

In the crushed area at the level of the fourth lumbar segment the vessels were found to be all thrombosed, the smallest capillaries being engorged with blood. There was also a large extravasation of blood outside the cord, and the crushed substance of the spinal cord had been squeezed upwards (*i.e.*, headwards) beneath the arachnoid for two segments distance, so as to simulate the heterotopic appearance of a double cord.

[1] We would support the use of the expression fasciculus spino-tectalis for this tract as being shorter and more in accord with general morphological terminology.

Fig. 1


Cervical Cord.
(Man)
Note with less large number of scattered direct spino-bulbar fibres
crossed pyramidal degeneration
Spino vestibular and dorsal spino-cerebellar fibres
sharply defined area of central spino cerebellar & spino-tectal & Spinothalamic fibres
ascending ground bundle fibres lying in form. ret.
ascending fibres of posterior longitudinal bundle

Dr. E. J. Worrall, *Phot.*

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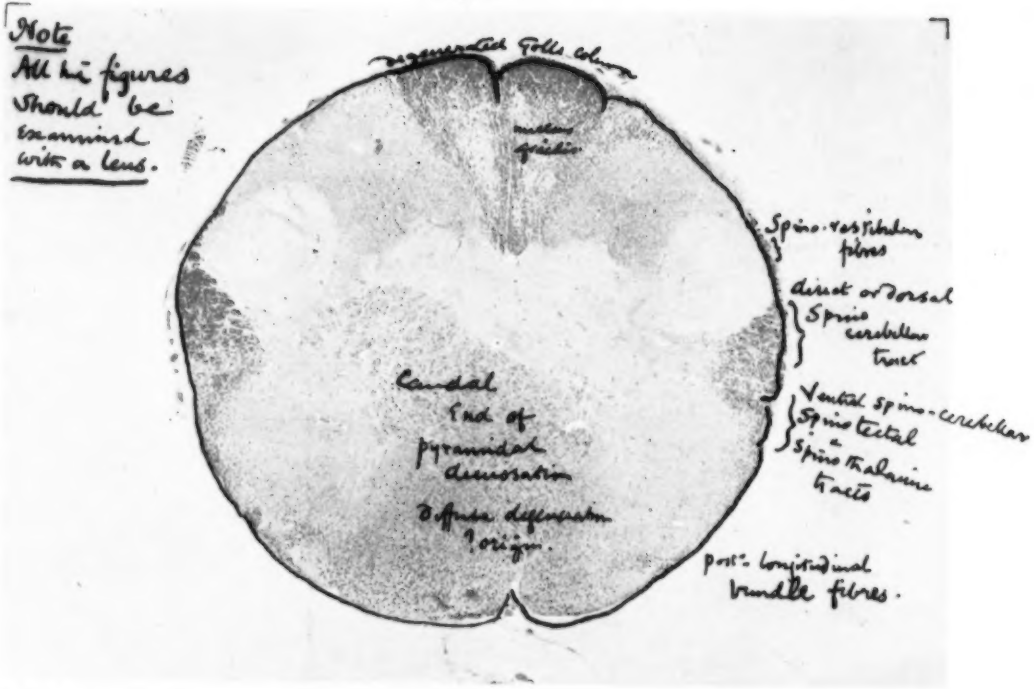
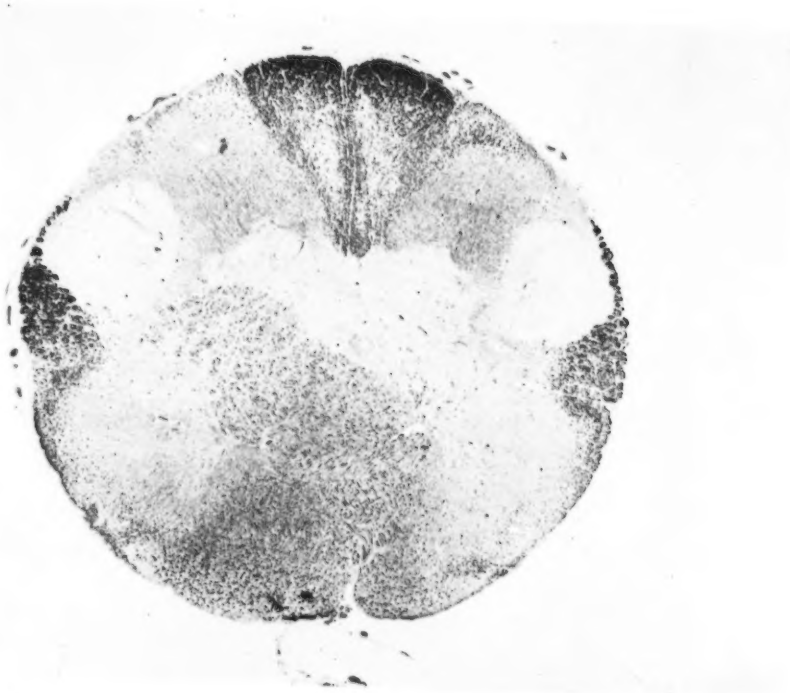
Fig. 2


Note
All the figures
should be
examined
with a lens.
Degenerated Colls of
Goll's
Spino-vestibular
fibres
direct or Dorsal
Spino
cerebellar
tract
Ventral Spino-cerebellar
Spino tectal
&
Spino thalamic
tracts
Caudal
end of
pyramidal
decussation
Diffuse degeneration
? origin.
post. longitudinal
bundle fibres.



Dr. E. J. Worrall, *Phot.*

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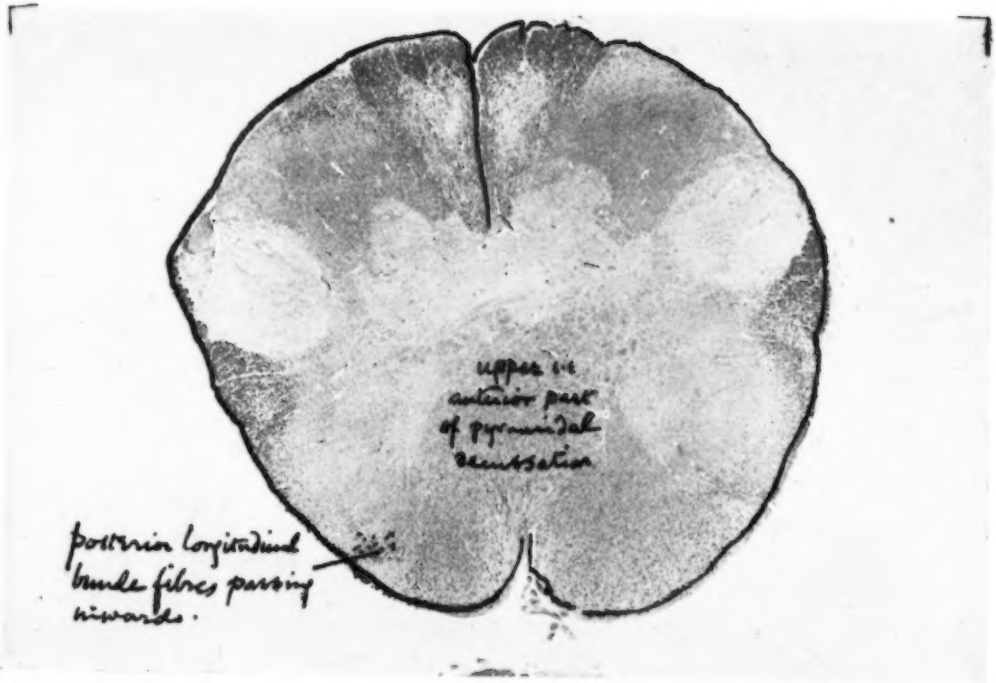
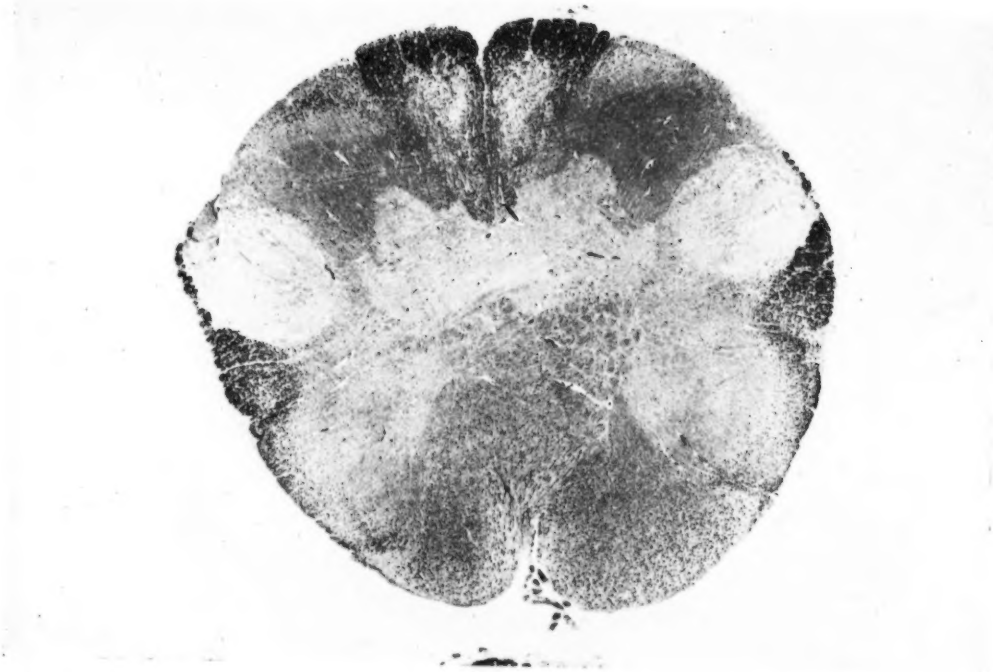
Fig. 3



Dr. E. J. Worrall, *Phot.*

Taylor & Danielsson, Ltd., London.

Fig 3



Dr. E. J. Worrall, *Phot.*

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(1) *The Direct Cerebellar Tract of Flechsig or the Fasciculus Spino-cerebellaris Dorso-lateralis.* (*Barker.*)

This tract is seen in the lowest sections as a bundle of coarse fibres symmetrically degenerated on either side of the cord. It has its customary situation, and extends forward to the ligamentum denticulatum, at which point it is continuous with the antero-lateral tract. At the level of the decussation of the pyramids the degeneration reaches dorsally to the inner border of the subst. gelatinosa on the one side but not on the other; it maintains this relation at the middle of the pyramidal decussation. When the restiform body is fully formed the direct cerebellar fibres occupy its anterior portion. In sections through the lower part of the bulb there are some coarse fibres to be seen lying ventrally to the spinal fifth root; these subsequently pass inwards and dorsalwards, and on examination prove to be only fibres of the direct cerebellar tract taking this somewhat unusual course. The coarse direct cerebellar tract fibres run straight upwards and backwards into the white matter of the cerebellum accompanied on each side by a broad band of fibres which are not degenerated, and which form the olivary division of the restiform body. These direct cerebellar fibres arrive opposite the lateral edge of the lowest folium of the vermis and there branch and subdivide. A few pass under the border of the vermis, but the large majority pass dorsally and spread out into the white matter, and ultimately pass across in the anterior commissure. As the fibres pass backwards they are so arranged that the olivary constituents of the restiform body run caudal to the spinal fibres. As they enter the cerebellum some degenerated fibres can be traced directly to the peduncle of the flocculus.

In the posterior commissure of the cerebellum only half the fibres therein are degenerated, the other (non-degenerated) half are therefore probably olivary in origin.

The direct spino-cerebellar fibres terminate mostly in the superior vermis, and are distributed in diminishing degree in the rest of the cortex cerebelli of each side, the fibres

diminishing in number and calibre, so that the fibres in the posterior superior lobule are extremely fine, whereas those in the superior vermis are both fine and coarse.

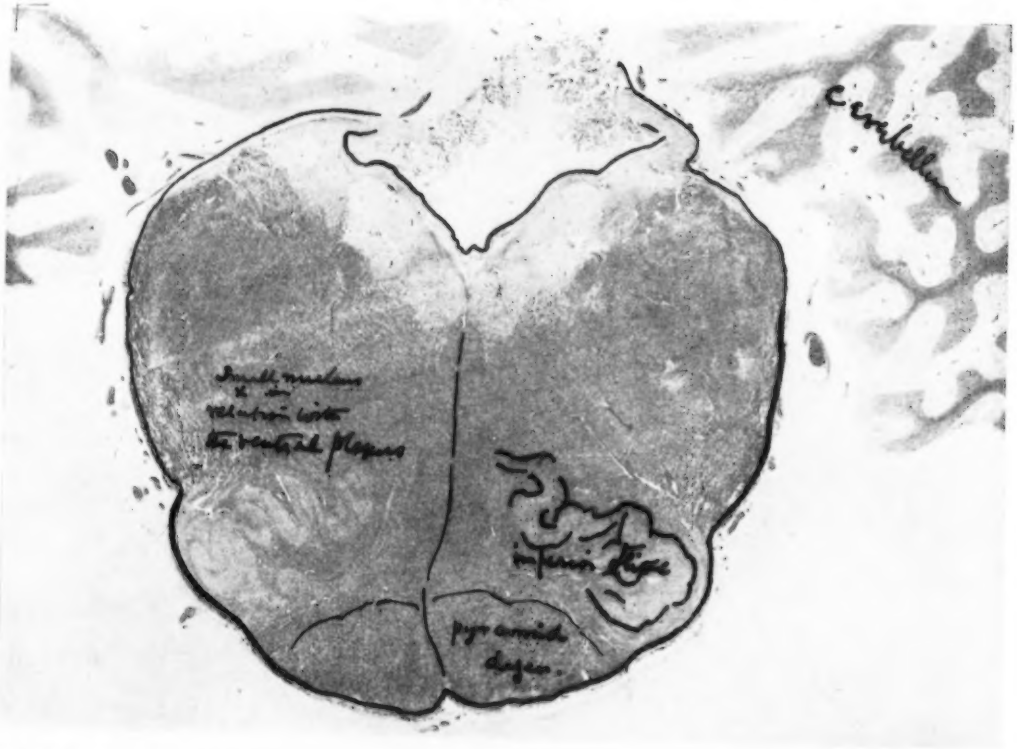
The distribution of degenerated fibres to the cerebellar cortex is most marked in the ventral division of the lateral lobes.

So far the arrangement of the fibres observed corresponds with that usually described. We wish now to draw attention to the special relations of the direct spino-cerebellar tract to the structures in the lateral region of the bulb and termination of collaterals derived from the tract. These we will describe according to their morphological appearance, which is that of plexuses.

Fine collateral plexuses in and round the spinal root of trigeminus.—(*a*) *Ventral collateral plexus.* Just above the uppermost level of the decussation of the pyramids strong collaterals springing from the fibres of the direct cerebellar tract as they turn up towards the restiform body pass inwards just ventrally to the spinal trigeminus root and between that and the antero-lateral nucleus. This may be termed the ventral bulbar collateral plexus of the direct cerebellar tract. The fibres ultimately composing it are very fine, and appear to terminate in relation with a nucleus of small nerve corpuscles lying in a plane just internal to the ventral border of the spinal trigeminus root. The uppermost limit of this ventral plexus is the upper border of the auditory striæ. We have found a definite interruption in this plexus for some distance (from 1 to 2 mm.), and that it consequently reappears soon before it finally ends. The nucleus referred to reaches in the formatio reticularis from 1 to 2 mm. higher than the end of the plexus, and is, perhaps, the posterior subdivision of the nucleus lateralis.

(*b*) *Dorsal collateral plexus.*—Another plexus of fine collateral (?) fibres is seen to come from the direct cerebellar fibres, passing inwards and partly through the spinal fifth root. These are collected in a fine plexus just external to the gray nucleus enveloping the fasciculus solitarius, and is, of necessity, especially well defined on the outer side of the gray matter surrounding the fasciculus solitarius. It

Fig. 4



Dr. E. J. Worrall, *Phot.*

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Dr. E. J. Worrall, *Phot.*

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Fig. 5

Fig. 5

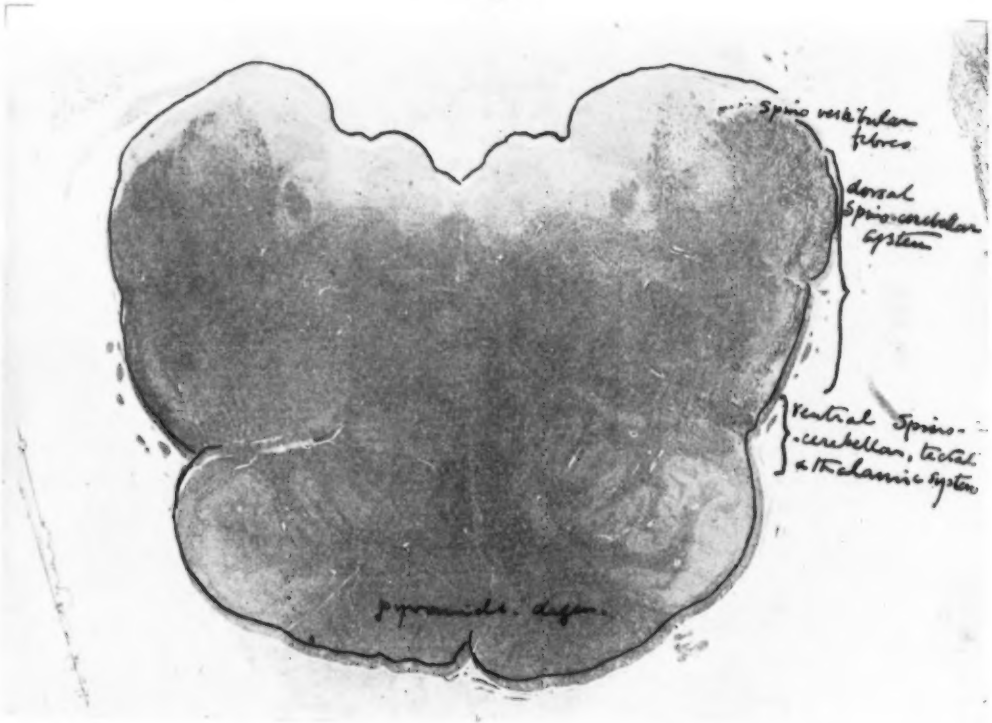
Spino vestibular fibres
dorsal Spino-cerebellar system
Ventral Spino-cerebellar, tectal & Thalamic system
pyramids. degn.

Fig. 5

Dr. E. J. Worrall, *Phot.*

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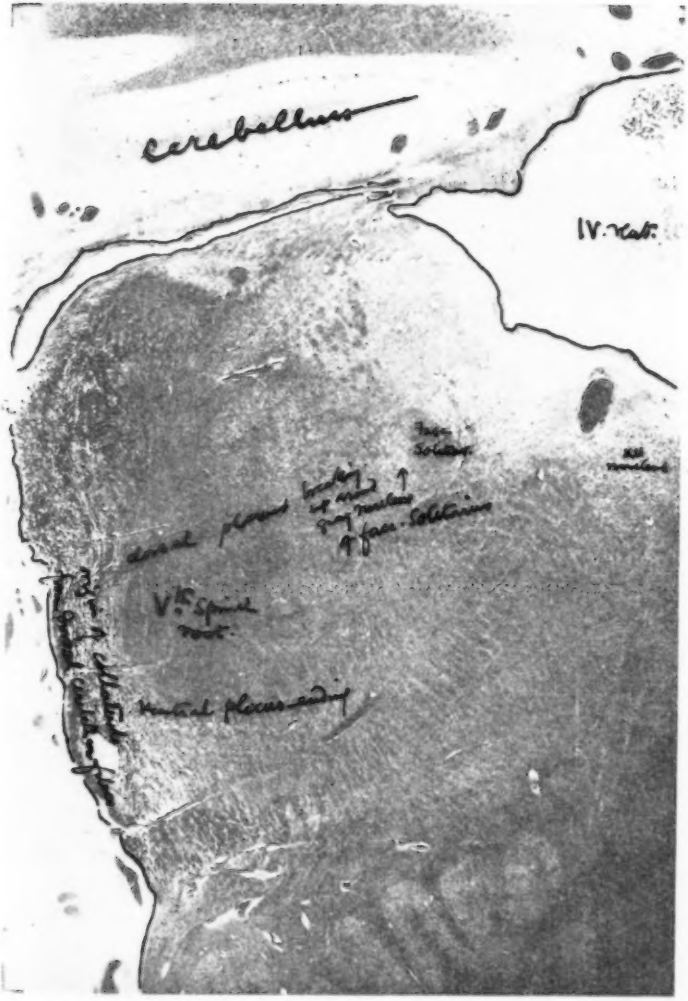
Fig. 6



Bale & Danielsson, Ltd., London.

Fig. 6

Dr. E. J. Worrall, *Phot.* 6 *Bale & Danielsson, Ltd., London.*

commences below in a plane close above the highest level of the pyramidal decussation. At the level of the upper end of the inferior olive the fibres of the dorsal plexus pass towards the central gray matter, and the plexus receives but few contributory fibres from the external arcuate fibres. The upper limit of this dorsal plexus is the highest level of the auditory striæ.

These fine plexuses, which in our preparations arise directly from the external arcuate fibres, were first noticed by Hoche in a case of transverse lesion of the spinal cord at the level of the seventh thoracic segment (*Archiv. für Psychiat., Bd.* 28, 1896), and Hoche's drawings correspond very closely with our photographs.

It has been suggested, however, by Barker that these plexuses are the termini of spinal fibres ascending through the posterior column nuclei. As already stated, however, we have proved their origin to be from collaterals which arise from the external arcuate fibres.

But for the fact of the marked association in physiological activity between the vagus phenomena of vomiting and nausea and disturbances of equilibration it would be difficult to explain the close connection of the spino-cerebellar system with the nuclei of the vagus and glosso-pharyngeal nerves such as is provided by these plexuses.

(*c*) *Spino-vestibular fibres.*—As constituting a separate group of external arcuate fibres, we wish to draw attention to a well-marked bundle of medium-sized fibres which pass outside the thin layer of gray matter which covers the restiform body. These fibres curve round the restiform body and pass ventrally to the descending root of the vestibular nerve as well as towards the plexus just described as lying outside the fasciculus solitarius. Consequently there exists a spino-vestibular system which runs with the most dorsal direct spino-cerebellar fibres.

(2) *Gowers's Tract or the Fasciculus Spino-cerebellaris Ventralis. (Barker.)*

As the researches of Mott have shown that what was known as Gowers's or the ventro-lateral tract is really a

complex of several systems, we have endeavoured to separate these systems, and, as stated above, regard them, at any rate, to be so far determined as to permit of their being enumerated as spino-cerebellar, spino-quadrigeminal, and spino-thalamic fibres, respectively.

It appears to us from examination of the present case that there is also another system included among the fibres of the ventro-lateral tract, and that is a small system of finer fibres which apparently terminate in the nucleus of of the lateral fillet.

To deal with these points successively and systematically we venture to propose to make use of the expression Gowers's tract only in reference to the system of spino-cerebellar fibres.

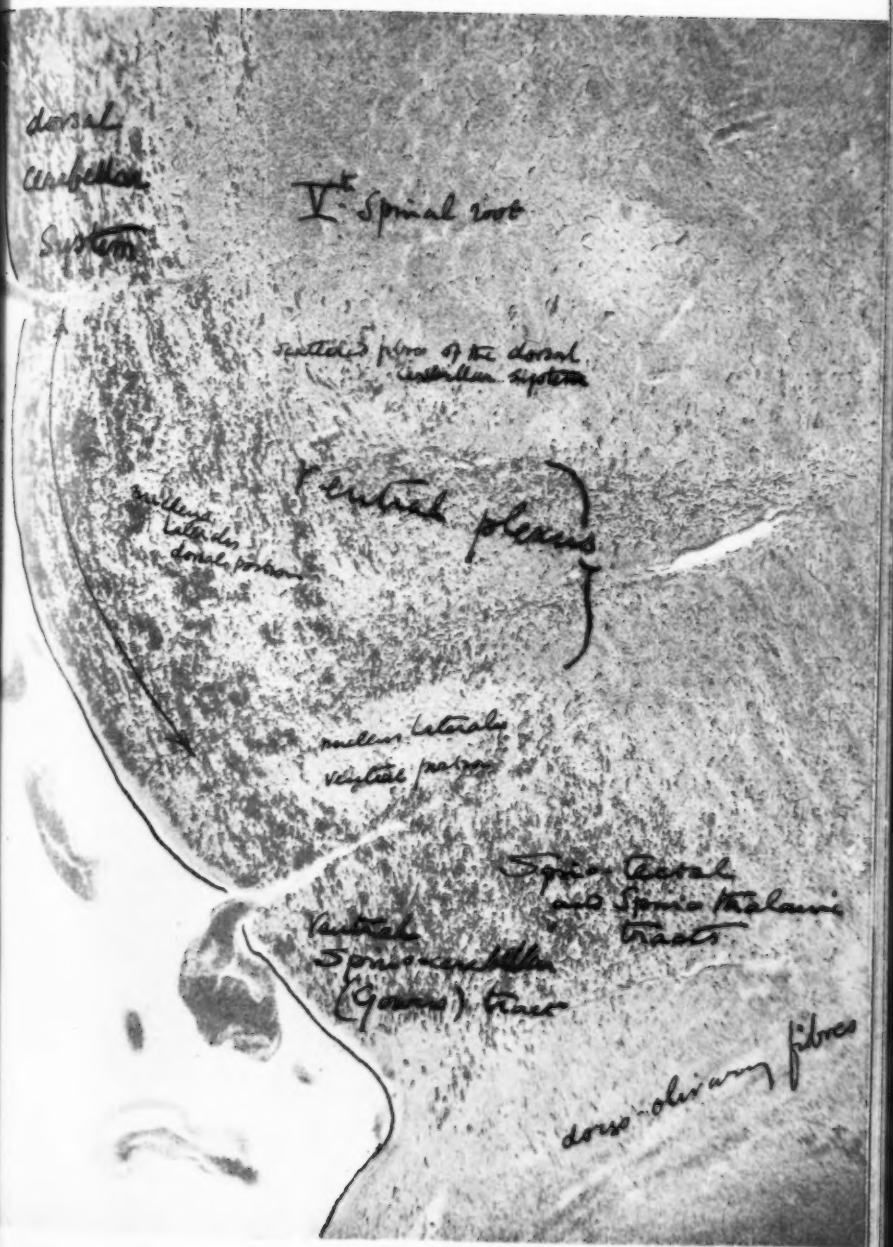
We would point out prefatorily that the ventro-lateral field in the spinal cord shows a difference in the size of the fibres; that the outer fibres are rather coarser than the inner division, and further, that this appears to afford a basis for recognising the different tracts from each other. Thus in the higher sections up to (and including) the pulvinar region the coarser fibres pass by way of the superior peduncle to the cerebellum, but the finer fibres can be detected passing as spino-thalamic and as spino-quadrigeminal tracts.

The fibres of Gowers's tract proceeding to the cerebellum do so compactly as a thick bundle just outside the sheet of gray matter (continuous really with the central gray substance), which laps over and round the superior cerebellar peduncle. As the tract lies on the outer surface of the cerebellar peduncle it is intercalated as a layer between two other systems of fibres which are as yet undetermined, and on the highest point it is covered with a thin layer of sub-pial gray substance.

Owing to the fact that in the present case both tracts are completely degenerated, as well as the dorsal spino-cerebellar system, we are not able to add to the present knowledge of the termination of Gowers's tract in the anterior portion of the vermis.

At the same time we wish to draw attention to the following groups of fibres which bear a relation to the ventro-lateral spino-cerebellar system.

Fig. 7



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Of these we would note first that fibres leave the tract, enter the velum medullare anterius and pass by way of the frenulum of the posterior corpora quadrigemina to the colliculus of either side.

The second group is much more important and difficult of analysis. Thus it is easy to trace fibres from one tract passing across the roof of the fourth ventricle to the opposite side, where they appear to descend in the sub-ependymal tissue towards the peduncle of the flocculus and the nuclei of the eighth nerve.

We must now discuss previous observations which bear on this group of fibres.

In the rhomboidal area bounded by Deiters's nucleus below, the bundles of the restiform body externally, the lateral wall of the fourth ventricle internally, and the brachium conjunctivum above, there have been for many years observed fibres extending upwards from the region of Deiters's nucleus and the embolus, and to the nucleus tecti in the cerebellum. These fibres are fine fibres, and have been variously described by different observers.

Thus Stilling (p. 175, table xv., *Stilling Disqu. de Struct. et Funct. Cerebri*) spoke of them as being the continuation of the column of the fasciculus cuneatus and the fasciculus gracilis. Bruce pointed out in 1892 that many of them came from the neighbourhood of the superior olive. Edinger, a few years later, showed that they were posteriorly apparently the continuations of various sensory cranial nerves, and he spoke of these bundles as a direct sensory cerebellar tract.

Kölliker describes them (p. 270, *Handbuch der Gewebelehre*) as a strong bundle passing dorsalwards on the mesial side of the restiform body. He traced them to the nucleus tecti, and partly through the embolus and globosus, finding that after decussating they terminated in the nucleus tecti of the opposite side. Thus Kölliker adopts the prevalent view that these fibres are such as leave the neighbourhood of Deiters's nucleus to go towards the cerebellum, and says that another origin to these fibres is not obvious. At the same time he makes the most important observation that he

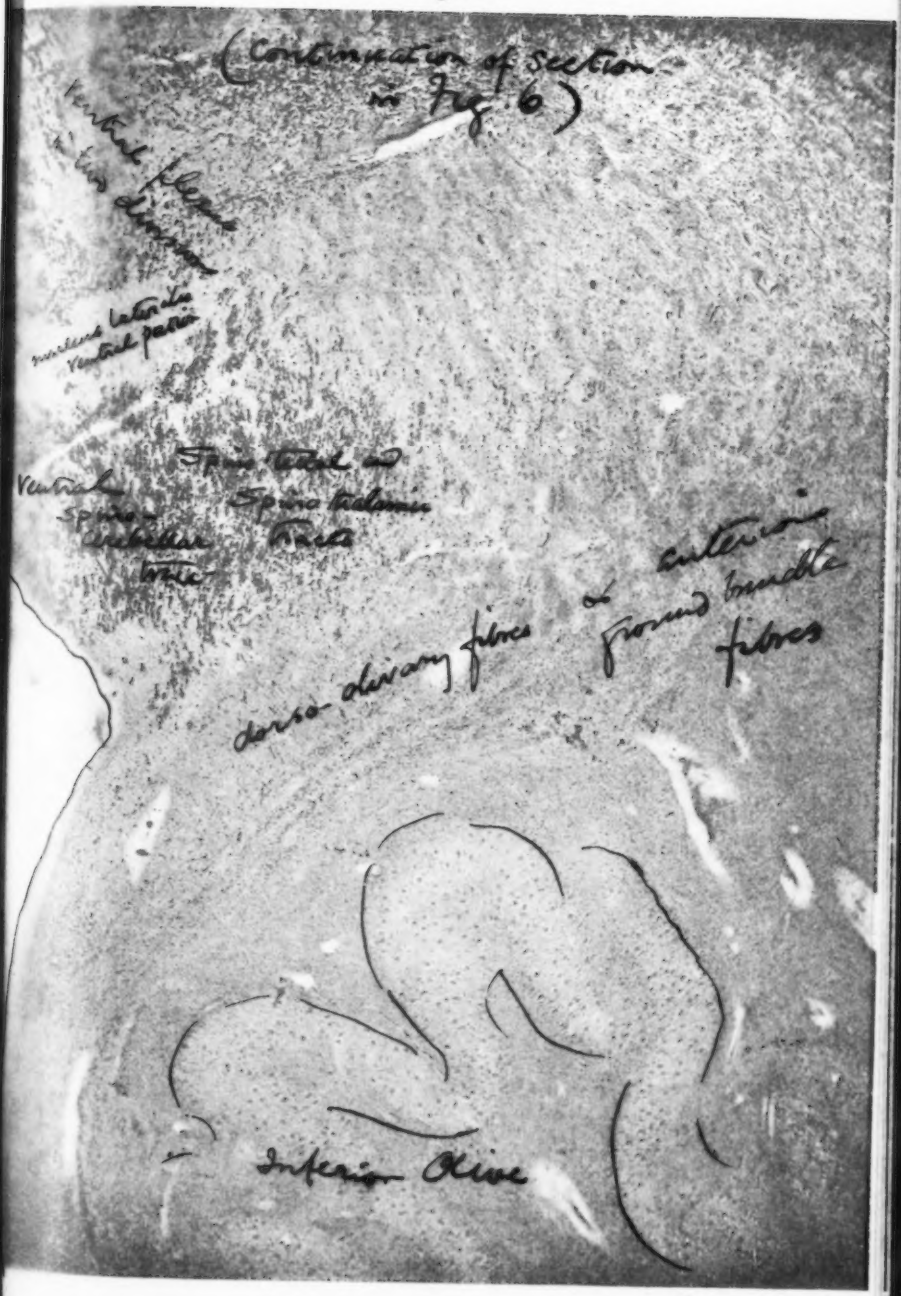
noticed in a human fœtus of the seventh to the eighth month the terminations of these fibres in the nucleus tecti to be already myelinated, whereas the "bundles of origin" were not, and thus contrasted very strongly with the medullated fibres of the nervus vestibuli and pedunculus cerebelli. This fact seems to us to render it at least improbable that the fibres ascend from Deiters's nucleus and the vestibular nerve into the cerebellum. In speaking of the region of these "bundles of origin," it should be noted that Bruce in 1892 showed the peduncle of the flocculus to spring from the area to which reference has been made. In the present case these fibres are degenerated, and have been described above as being possibly derivatives of Gowers's tract. In remarkable agreement with Kölliker's observation regarding myelination, we may again draw attention to the fact that in our case the lower ends of the fibres, where they are apparently connected with the upper part of Deiters's nucleus are distinctly branching and breaking up.

Our conclusions would be, therefore, that these fibres certainly come at any rate from the neighbourhood of the roof nucleus and pass down the side of the ventricle, to terminate in the upper part of Deiters's nucleus. Whether we are right in regarding them as being the continuation of the antero-lateral tract will have to be tested by subsequent observation, but in support of it may be mentioned that one of us has in the cat traced degenerated fibres of Gowers's tract across the roof of the fourth ventricle to the interval between the brachium conjunctivum and the lateral wall of the ventricle on the opposite side.

These same fibres are described by Miss Sabin in two papers, the most recent of which is that relating to her model of the medulla ("Contributions to the Science of Medicine, Dedicated to Wm. H. Welch, 1900," p. 990). In this connection she draws attention to two bundles of fibres connecting the vestibular nuclei with the cerebellum, which she calls inner and outer group respectively, of the fibres connecting the vestibular area with the cerebellar. Of these the mesial bundle passes into the roof.

The whole question of these fibres is a very difficult one

Fig. 8


(Continuation of section in Fig. 6)
ventral fibres of tract of Monakow
nucleus lateralis ventralis pars
Spino-tectal and Spino-thalamic tracts
Ventral Spino-cerebellar tract
dorso-olivary fibres of anterior ground bundle fibres
Inferior Olive

Hazell, Phot. Bale & Danielsson, Ltd., London

E. J. Wor

to solve, and with reference now to the question of Edinger's views regarding the direct connection of the vestibular nerve with the cerebellum, Ramon y Cajal (*Beitrag zm. Studium der Med. Oblongata, Deutsch. von Bresler*, Leipzig, 1896) includes in his fig. 18 fibres of the vestibular nerve, which, as they pass round the upper end of the spinal trigeminus root, bifurcate, the dorsal limb of the bifurcation running upwards, giving off collaterals to Deiters's nucleus, and terminating in the nucleus tecti. If this arrangement which is found in the mouse is confirmed for other animals and man, then some of these fine fibres must be taken as direct continuations of the vestibular nerve. Such a fact of course would not alter our view of the fibres observed in the present case, because such direct vestibular fibres would necessarily not be degenerated in our case.

Degeneration of the fleece fibres.—The connections of the corpus dentatum by means of the plexus surrounding it and known as the fleece (Stilling) are very important. In the present case numerous very fine degenerated fibres entered the plexus in large numbers on the dorsum of the corpus dentatum, but very few at the lower third of the ventral aspect of the nucleus.

As regards position in respect of the cerebellum as a whole it should be observed that the contributions the fleece receives from the restiform body enter its dorsal portion alone, so far as we were able to ascertain.

(3) *The Fasciculus Spino-tectalis vel Quadrigeminalis.*

This includes those fibres which terminate in the posterior and anterior colliculus respectively.

(*a*) *Fibres to the posterior colliculus.*—We have already stated that fibres leave the spino-cerebellar tract to enter the posterior colliculus by way of its frenulum. The main mass of fibres, however, to the posterior colliculus come towards its nucleus in association with the fibres which we find to end in the nucleus of the lateral fillet, and are running mesially to the latter. The spino-tectal bundles diverge beneath the nucleus of the posterior colliculus, forming a cup or funnel-shaped bed for the oval nucleus.

This description of the fibres beneath the ovoid nuclear centre of the posterior colliculus has been given by various anatomists for the fibres of the lateral fillet, and the spinal fibres are in the normal case not distinguishable from those of the lateral fillet, but are necessarily more caudal. In fact, the account given by Kölliker of the normal lateral fillet is exactly applicable to our spino-tectal (posterior) system.

(*b*) *Fibres to anterior colliculus.*—The termination of the spino-tectal system in the anterior corpus quadrigeminum is very definite. The fibres are at the most distal plane of the colliculus arranged in an oval bundle situated in the outer part of the deep medullary stratum, and therefore close beneath the posterior brachium. From this point the fibres ascending parallel to the longitudinal axis of the mesencephalon give off collaterals or turn off directly to run in the commissure between the corpora and in the deep medullary stratum. These give off collaterals at right angles, which enter the upper gray layers of the homolateral colliculus. Others pass across the middle line and enter the contralateral colliculus.

The gray layers of the corpus quadrigeminum thus receive the spinal fibres as follows: Those which end homolaterally pass into the outer part of the gray layers, and those which end contralaterally enter the inner portions of the gray layers.

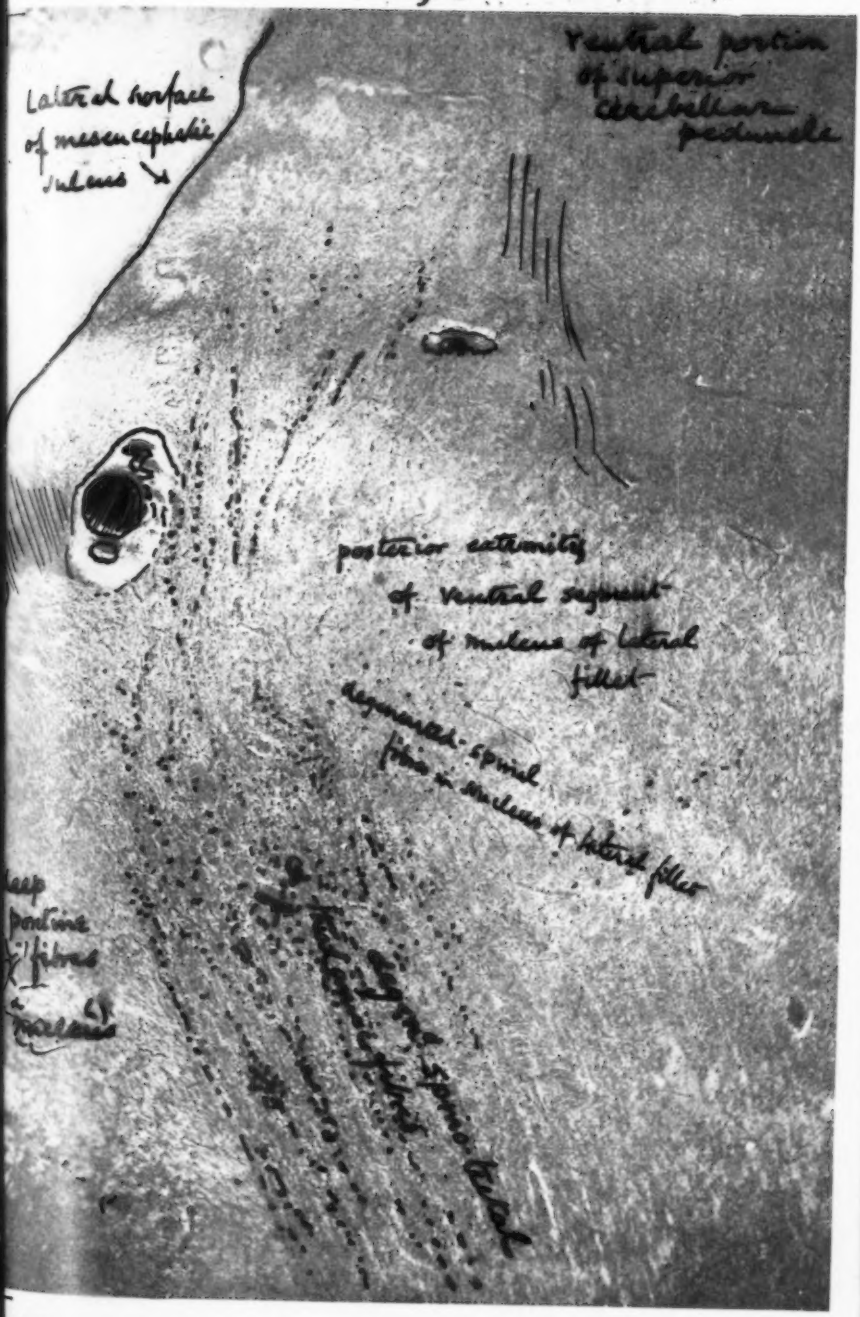
(4) *The Fasciculus Spino-thalamicus.*

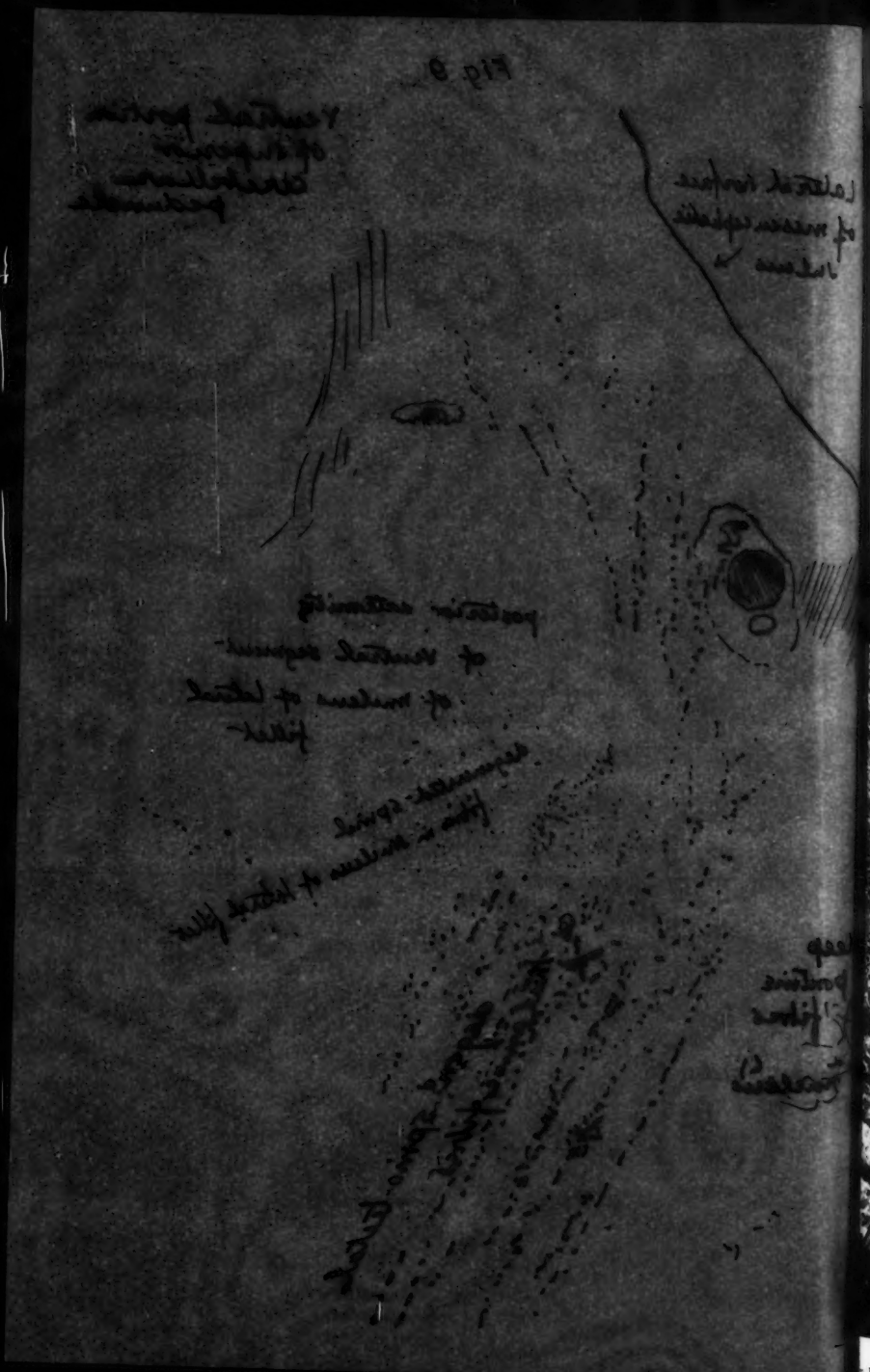
After the ascending ventro-lateral tract has given off its contributions to the cerebellum and corpora quadrigemina as just detailed, the remnant of the fibres pass on to enter the thalamus.

In doing this the fibres spread out so that the bundle presents in transverse section a V-shaped figure. These fibres enter the pulvinar, and constitute, doubtless, a tract of considerable phylogenetic importance.

A few words may be added concerning degeneration in tracts other than those described.

Fig. 9


Lateral surface of mesencephalic nucleus
Ventral portion of superior cerebellar pedunncle
posterior extremity of ventral segment of nucleus of lateral fillet
Segmented spinal fibres in nucleus of lateral fillet
deep pontine fibres in nucleus



E. J. WORSALL, *Phot.*

Fig. 10

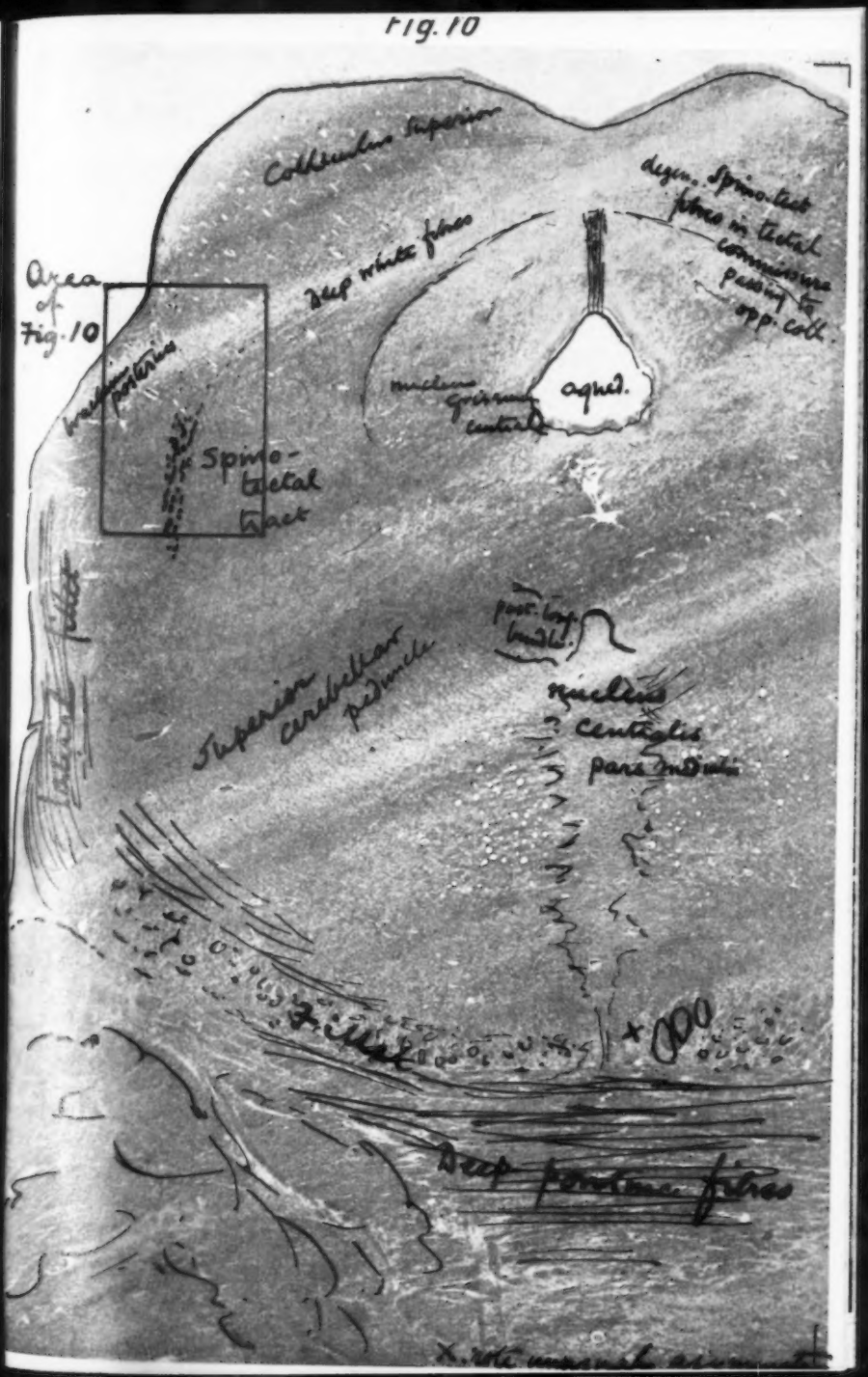

Colliculus superior
Area of Fig. 10
Deep white fibres
degen. Spino-tect fibres in tectal commissure passing to opp. coll.
aqued.
Spino-tectal tract
Superior cerebellar peduncle
Deep tectum. fibres

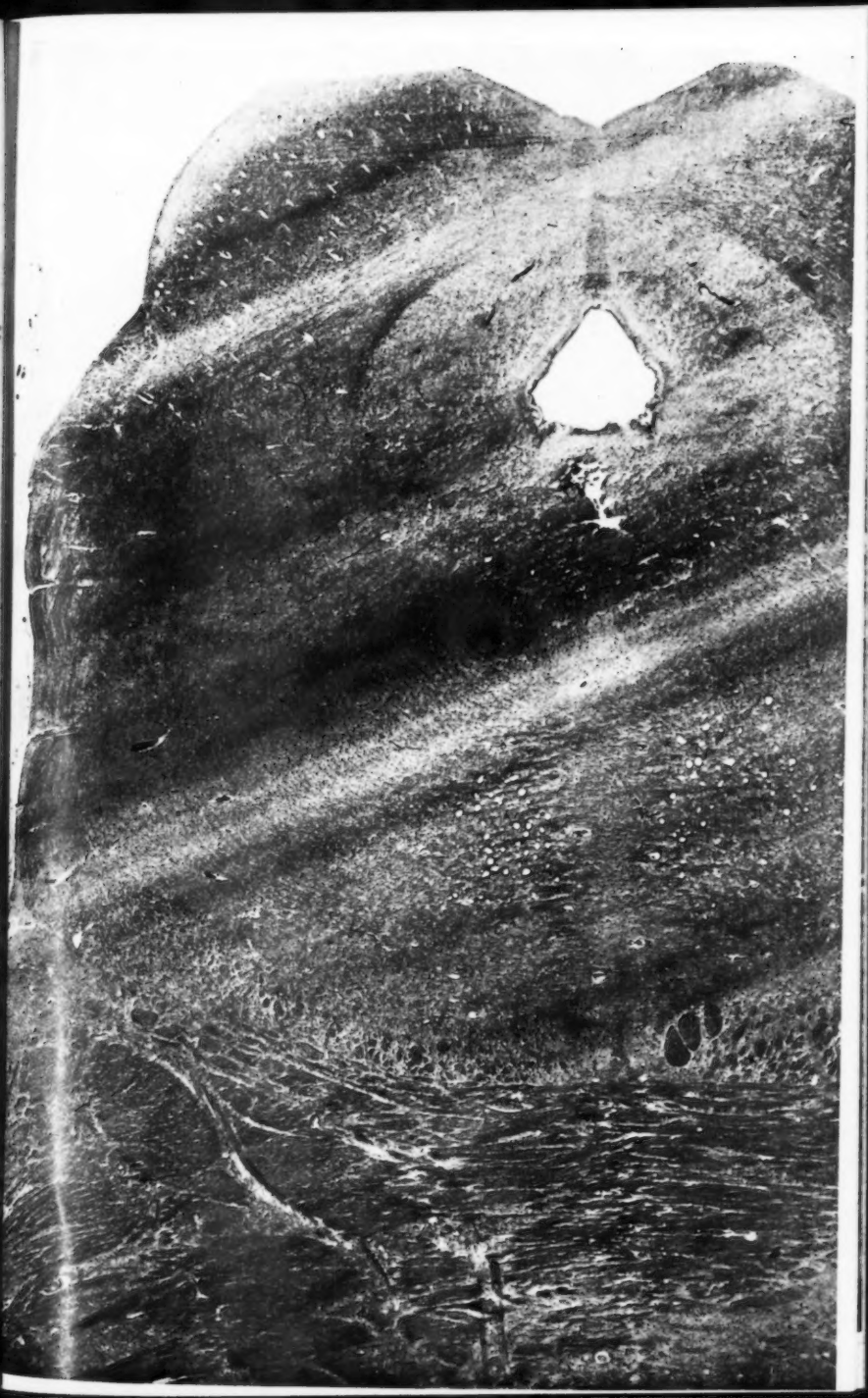
Fig. 10

Area of Fig. 10

Deep water flora

Spino-littoral zone

Deep water flora



(1) *Pyramidal system.*—A general and equal degeneration of coarse fibres was present in both pyramidal tracts, and in the spinal cord was spread over both the crossed tract and also the direct tract. The question whether this degeneration is possibly an ascending one has been raised by Dr. Purves Stewart in his case of injury of the cervical cord. In our case, however, sections across the parietal lobe showed not only extensive degeneration of large fibres descending (?) from the cortex, but also numerous medium-sized fibres passing into and across the corpus callosum.

Although no gross lesion of the cortex was discoverable in the osmic stained sections, we think that the above facts show that, probably as a result of concussion, the pyramidal corpuscles in the cortex suffered damage, and that their axones had degenerated in consequence. The patient in the present case undoubtedly received a severe shock, and, as mentioned above, suffered from a large scalp wound in the occipital region.

Pending further observations on this point it would perhaps be safer to regard the coarse degeneration of the pyramidal system as being of cortical origin and descending character.

Posterior longitudinal bundle system.—A few coarse fibres were traceable throughout the whole course of the posterior longitudinal bundle system. Thus in the anterior columns of the cord these fibres lay in front of the anterior commissure, and mingled ventrally with those of the direct pyramidal tract, occupying the mesial portion of the column. Higher up, at the level of the commencing decussatio lemniscorum, these same fibres lay close outside the fillet fibres. In their course up the medulla they kept close to the raphe, and finally in the mesencephalon formed the mesial border of the posterior longitudinal bundle. They began to disappear opposite the anterior corpus quadrigeminum, and at the commencement of the commissura mollis were reduced to a few units, appearing to end in the so-called nucleus of the posterior longitudinal bundle.

We have described these fibres as ascending in character because of their mode of termination and gradual disappearance headwards.

The "accessory fillet" (Schlesinger).—This is a tract which is in the main composed of medium-sized fibres. In the upper sections the fibres were compactly arranged and formed a tract, the fibres of which, passing downwards and outwards, became lost externally in the lateral fillet, and mesially in the tegmentum.

The tract first appears in the plane of the pulvinar. We believe with Redlich and Hoche that it is a downward efferent path of the pyramidal system, from which it obviously arises while traversing the pons.

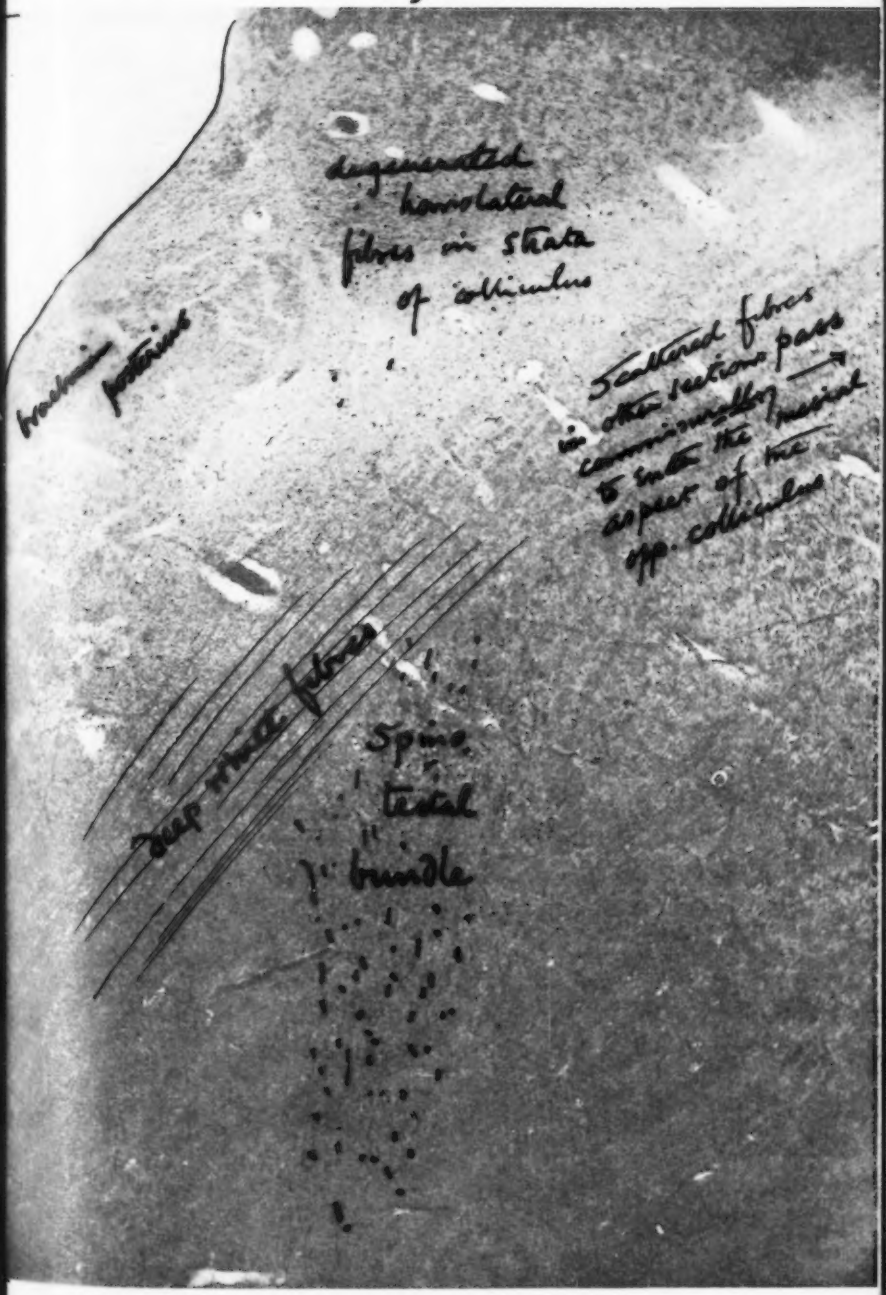
The dorso-olivary fibres (von Solder).—At the commencement of the hinder pole of the olive von Solder's tract was visible as numerous scattered, fine, degenerate fibres and a few coarse ones, which latter were continuous with those from the anterior columns left after the decussation of the pyramids is established. The fine fibres are marked all along the dorsum of the olive, and limited, dorsally, partly by the ganglion cells of the formatio reticularis, while laterally they were continuous with the medium-sized fibres of the antero-lateral tract. Some fibres were scattered around the dorsal accessory olive, reaching outwards ventrally to the ventral nucleus of the funiculus lateralis and coursed round the surface of the bulb, just outside the capsule of the outer end of the inferior olive.

Some of the most mesial fibres passed into the inner side of the inferior olive.

As the fibres were traced upwards they almost all passed above the outer part of the fillet and entered the tegmentum. In this way they passed straight up to the thalamus and terminated by subdividing minutely in the ventral nucleus of the thalamus. von Solder traced the same fibres as far as the plane of the corpus geniculatum mesiale.

Posterior column system.—In the cord Goll's columns were equally degenerated on the two sides, the degeneration reaching only about three-fifths of the way along the median fissure. On the one side Burdach's column is affected in its median fourth, and for only two-thirds of its length posteriorly. The degenerated fibres could be traced upwards to the level of the pyramidal decussation, the nucleus gracilis

Fig. 11


degenerated homolateral fibres in strata of colliculus
brachium posterius
Scattered fibres in other sections pass commissurally to enter the aqueduct of the opp. colliculus
deep arcuate fibres
Spino tectal bundle

Dale & Danielsson, Ltd., London.

E. J. WORRALL, *Phot.* 11 *Bale & Danielsson, Ltd., London.*

being there hardly marked, and at the same point the degenerated fibres became much finer.

At the lowest level of the bulb, where the corp. restiforme is distinctly formed, fine degenerated fibres, continuous with those of the posterior columns, form a delicate area of degeneration at the inner side of the restiform body, being separated from the degenerated spinal fibres by olivary fibres. These fine fibres join a fine meshwork of fibres in the dorsal part of the spino-vestibular root and nucleus, and appear to lose themselves in the gray matter just above the tuberculum acusticum, becoming finally lost beneath the ependyma as high up as the level of Deiters's nucleus.

The nuclei of the posterior columns show extensive degeneration, and from them a few fine degenerate fibres pass down as internal arcuate fibres into the fillet of each side. Similar fibres were first observed by Purves Stewart, but they disappear very soon from the fillet, and resemble fine collaterals rather than direct fibres.

Degeneration of the stria medullaris.—The fibres of the stria entering the ganglion habenulæ were in this instance degenerated, as also some coarse fibres in the posterior commissure. We were, however, unable to determine the cause or relations of these degenerations.

NOTE.—I desire to add to the above account the fact that the heavy labour of preparing the large series of preparations in the above case was executed by Dr. Thiele. —V. H.

THE OCCURRENCE OF OPTIC NEURITIS IN LESIONS OF THE SPINAL CORD. INJURY, TUMOUR, MYELITIS. (AN ACCOUNT OF TWELVE CASES AND ONE AUTOPSY.)

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CONSIDERING the rarity of recorded cases in which optic neuritis has been associated with lesions of the spinal cord other than cerebro-spinal meningitis, we venture to think that a series of twelve cases which have lately come under our observation are worthy of being recorded, especially since in one case a pathological investigation was made by one of us (J. T.). Our best thanks are due to Dr. Hughlings Jackson, Dr. Buzzard, Dr. Bastian, Sir William Gowers and Dr. Ferrier for permission to record cases under their care, and to Mr. Brudenell Carter and Mr. Marcus Gunn for permission to use their valuable notes upon these cases.

This paper contains :—

(1) An account of twelve cases.

The detailed clinical account of these cases will be found in the appendix at the end of this communication.

(2) A list of all the recorded cases we have been able to find in the literature of the subject.

(3) An analysis of all the cases of local lesions of the spinal cord which have been treated at the National Hospital during the years 1892-1900, with reference to the state of the optic discs.

Clinical Cases.

(*A detailed account of these cases will be found on p.* 541.)

Case 1.—*Tumour at the Third Cervical Segment.*

Female, aged 56 years. Slowly increasing spastic paraplegia and anæsthesia involving all four limbs. Marked weakness of muscles of neck. Well-marked double optic neuritis. Death two months later. At the autopsy an extra-medullary intrathecal myxoma, about the size of a sparrow's egg, was found pressing into the cord at the level of the third cervical segment. There was no growth in the brain and no meningitis present.

Case 2.—*Tumour at the Fifth Cervical Segment.*
(? + *Cerebral Tumour.*)

Male, aged 34 years. Syphilis at 27 years. Since 29 years periodic epileptic attacks, preceded by intense headache. Gradual onset of paraplegia for five months. Absolute flaccid paraplegia in all four limbs. Absolute anæsthesia everywhere below the fifth cervical area, except over lumbar 5 L., where all forms of sensibility were much impaired. Much wasting and loss of faradic excitability in muscles supplied from fifth and sixth cervical segments. Well-marked double optic neuritis; swelling = $6\frac{1}{2}$ D. left eye and $4\frac{1}{2}$ D. right eye. Small hæmorrhages and effusions present in both discs. Six months later both discs presented well-marked post-neuritic atrophy, and vision was much impaired.

Case 3.—*? Tumour of Fifth Cervical Segment.*

Male, aged 48 years. Syphilis at 20. Gradually increasing spastic paraplegia of six months' duration, with shooting pains in the lower part of the neck. Left upper limb completely paralysed. The other limbs markedly paralysed. Complete analgesia and thermo-anæsthesia, with slight tactile impairment below the fourth cervical area. The edges of the optic discs were nowhere distinguishable.

There was exudation at the centre of the discs and along the vessels, with marked striation of the outer parts of both discs. There was no measurable swelling.

Case 4.—*Spinal Caries.*

Female, aged 14 years. Pain in cervical dorsal spine and gradually progressive spastic paraplegia of six months' duration. Paraplegia complete in three months; upper limit of anæsthesia one inch above the nipples. There was well-marked double optic neuritis, which increased markedly for the first fortnight of her stay in hospital, after which it gradually disappeared, leaving only slight blurring of the disc margin. There was an obsolete tubercle in one eye, and headache was present, but no vomiting. She was treated with rest and extension, and completely recovered from the paraplegia. At the present time (nine years later) she is in the best of health.

Case 5.—*Fracture Dislocation of Sixth Cervical Vertebra.*

Male, aged 28 years. As the result of a fall on shoulder, pain and paralysis of both arms and gradually on-coming spastic paraplegia. Upper limit of anæsthesia at lower limit of eighth cervical area. Marked optic neuritis in left eye developed while he was under observation. A piece of bone pressing on the cervical segment was removed by laminectomy. The optic neuritis rapidly subsided, leaving atrophy of the left disc and vision = $\frac{6}{24}$ and J. 14. He completely recovered, and at the present time (nine years after) he enjoys good health, having been at work ever since he left hospital.

Case 6.—*Acute Transverse Myelitis Seventh Dorsal Segment: Optic Discs Normal. A Second Attack Four Months Later in the Lower Cervical Region associated with Optic Neuritis.*

Male, aged 24. In March, 1899, he awoke one morning to find himself paralysed and numb from the waist downwards. The usual symptoms of transverse myelitis were

present. In July, 1899, while improving, slight optic neuritis was noticed. Four days later a rapid onset of paralysis of the hands, with complete anæsthesia below the third dorsal area, occurred. The neuritis increased rapidly, began to subside a fortnight later, and disappeared in four months, leaving obvious marginal blurring and connective tissue along the vessels. The symptoms of the cervical lesion improved greatly, those of the dorsal lesion did not improve.

Case 7.—*Acute Myelitis Upper Dorsal Region. Evidence of past Optic Neuritis.*

S. C., aged 36 years.—Rapid onset of paraplegia and anæsthesia, nine months before admission. Slight recovery of sensibility. In the right optic disc the edge was woolly and indistinct, the veins were distended and there was some connective tissue along the vessels. Some central filling and many fine vessels over the disc were present. There was no swelling. The left disc presented similar but less marked changes.

Case 8.—*Acute Myelitis, upper limit—Sensory change—the Second Dorsal Area.*

Male, aged 34 years. Paraplegia becoming complete in three weeks following an acute febrile illness, complete spastic paralysis (diaphragm excepted) below the fifth intercostal space. Upper extremities normal. Extreme hyperæsthesia round upper thorax (third to fifth rib). Thermo-anæsthesia as high as third intercostal space; pupils myotic.

Both optic discs were high coloured, and their edges were distinctly blurred though traceable, the retinal veins were overfull, appearances indicating an early stage of papillitis.

Case 9.—*Acute Myelitis, Second Dorsal Segment.*

Female, aged 22 years. Congenital syphilis. Acute onset of paraplegia and anæsthesia at twenty years old, followed by rapid failure of sight.

Both fundi showed advanced post-neuritic atrophy. Vision was reduced to light perception in the left eye, and to counting fingers at a distance of a foot with the left.

Case 10.—*Lesion of Cervical Cord, (?) Hæmatomyelia.*

Female, aged 15 years. Sudden loss of power and sensibility in arms, legs and trunk, accompanied by sharp pain between shoulders. No causal agent apparent.

Slight analgesia below fourth cervical area. Great atrophy and loss of faradic excitability in intrinsic hand muscles and in ulnar flexors. Double "main en griffe."

Absolute spastic paralysis of trunk and legs.

There was well marked optic neuritis in each eye, with several hæmorrhages near the centres of the discs.

The left pupil did not react to cocain, and the right only feebly.

Case 11.—*Chronic "Myelitis," upper limit at the Fifth Cervical Segment. Post-neuritic atrophy of both optic discs.*

Male, aged 41 years. Slow onset of spastic paraplegia for fifteen years. Slight anæsthesia to all forms below the fifth cervical root area. Both discs presented a blurred woolly indistinct margin with some connective tissue along the vessels, the latter being very small. Both were very pale and there was evidence of old exudation at the centre of each.

The condition of the discs was that typical of consecutive atrophy following a neuritis of moderate degree. It was first noticed by one of us (J. T.) two years before this patient's admission. No change had taken place in the appearance of the discs nor in the acuity of vision during this time.

Case 12.—(?) *Myelitis, Upper Dorsal Region.*

Male, aged 28 years.—Failure of vision associated with pain in dorsal spine, followed in a few weeks by onset of

spastic paraplegia. Double optic neuritis leaving some post-neuritic atrophy at the end of three months. Left Argyll Robertson pupil. Upper limit of anæsthesia at the sixth dorsal level. Great improvement in symptoms at the end of three months.

The most striking feature of the above cases is that, notwithstanding the varied nature of the lesions, myelitis, compression, injury and probably hæmorrhage, the situation in all of them the cervical, or the upper dorsal regions of the cord. An analysis of the recorded cases (p. 552) shows a similar result, and if those cases which have been verified *post mortem* be taken alone, the lesions are found to have been confined to even a narrower limit in the downward direction, for we have been unable to find a recorded case in which the lesion did not extend higher than the third dorsal segment. In making this statement we exclude certain recorded cases in which the clinical and the pathological results were widely at variance, as for instance in Devic's case, where he repeatedly found the upper limit of anæsthesia above the nipple, and at necropsy the only lesion found was in the lumbar enlargement. It is difficult to explain the origin of the statement that has found its way into several recent text-books of medicine that optic neuritis in spinal-cord disease occurs in association with disseminated myelitis, and that in the large proportion of the cases the myelitis is confined to the dorso-lumbar region. Neither among the published cases nor among the records of the National Hospital for the past ten years have we been able to find any case of spinal-cord lesion associated with optic neuritis in which the clinical records or pathological results proved the cervical enlargement of the spinal cord to be uninvolved (except perhaps our own case 12), and conversely. We suggest, therefore, that the occurrence of optic neuritis in these cases is dependent in part upon the situation of the lesion.

The nature of the lesion in the above cases is various, the larger proportion being examples of transverse myelitis,

which condition has been found in a large majority of the recorded cases. In three cases of disseminated myelitis which we have observed at the National Hospital, optic neuritis was not present, and in two only of the recorded cases was the existence of this lesion certain. A case of spinal tumour almost identical with the first of our series has been recently published by Nonne, and one of fracture dislocation of the vertebræ (C. 7, C. 8, D. 1, D. 2), resembling case 5, with a similar happy result by Firth. Several cases of spinal caries with slight changes in the optic discs are on record; Sir William Gowers states, however, that he is conversant with such cases, and that the changes are scarcely to be regarded as those characteristic of optic neuritis. We have not found optic neuritis of such degree as occurred in case 4 to have been recorded. The absolutely sudden onset of the spinal symptoms in case 10 rendered the diagnosis of hæmatomyelia very probable. Several cases in which this condition has been found *post mortem* will be found in the accompanying table.

In some of the recorded cases of myelitis associated with optic neuritis the disease has been narrowly limited, in others it has been of very wide extent, as, for example, in the case recorded by Mahokian and that by Schluster and Mendel, where the spinal cord was practically destroyed from the lower cervical to the sacral region. In about two-thirds of the cases of myelitis a history of syphilis has been obtained, and in relation with this fact it is noticeable that the incidence of the disease is chiefly in the third and fourth decades of life.

We have been unable to find among the records of the National Hospital any case in which optic neuritis was associated with syringomyelia. Neither was this sign present among a series of twenty-four fatal cases of sub-acute combined degeneration, notwithstanding the presence of severe anæmia in many of them, and that the disease involved the cervical region in the majority of them. In four, however, hæmorrhages were present in the retinæ.

We suggest, then, that the association of optic neuritis with a lesion of the spinal cord depends upon (1) the

position of the lesion; (2) some peculiarity in the nature of the disturbance of the functions of the spinal cord produced by the lesion, for in the recorded cases the damage to the cervical enlargement, although in some cases extensive, has been partial only.

The occurrence of unilateral optic neuritis, or of neuritis of marked difference in its degree in the two eyes, in cases in which symptoms pointed to a great affection of one lateral half of the spinal cord as in case 5, and in two other recorded cases, is interesting in this connection. When the relative frequency of the occurrence of optic neuritis is considered, the records of the National Hospital show that even when instances of lesions of the cervical region alone are analysed collectively, optic neuritis occurs only in a small percentage of the cases.

In the larger number of the recorded cases, as in our series, the neuritis has not been of severe degree. Such neuritis increasing for a few weeks after its appearance usually has commenced to subside within two months of its onset whatever is the cause of the condition producing it, sometimes disappearing so completely as to leave hardly a trace of its former presence; it does not usually lead to an incapacitating degree of atrophy and loss of vision. In several recorded cases, as in cases 6 and 12, the neuritis has preceded the appearance of paraplegia by some days or weeks.

In not a few cases, however, the most intense optic neuritis with extensive hæmorrhages has occurred, and amaurosis shortly following the occurrence of the lesion, and subsequently progressing to complete and permanent blindness, has called the early attention of the patient and physician to the eyes.

It is interesting to note that in several recorded cases the sudden occurrence of reflex iridoplegia while the patient was daily observed has been noticed, sometimes preceding, sometimes succeeding, the occurrence of the neuritis. The Argyll Robertson pupil has been in some cases permanent and in some temporary only. Myosis and inequality of the pupil have also been observed. While some of our cases

presented loss of the light reflex, this did not occur under our observation, neither, so far as we are aware, was the phenomenon transient.

We wish to bring forward the suggestion that, in addition to optic neuritis, headache, vomiting, or both, may occur in lesions of the cervical cord, especially in spinal tumours and pressure lesions. This association, which occurred in three of our cases, has been noticed also in other cases, and has led to the diagnosis of concomitant spinal and intracranial lesions. Yet subsequently the course of the disease or pathological investigation has rendered it probable or certain that such a double lesion had not existed. It is remarkable in this connection that, while cases of cerebral tumour presenting optic neuritis may run their course without either headache or vomiting, it is extremely rare for headache or vomiting to occur in those cases of intra-cranial growth which run their course without presenting optic neuritis.

We are unable to put forward any theory as to the causal relation of optic neuritis with lesions of the upper part of the spinal cord, but we hope at an early date to publish the results of experimental lesions upon the cervical cord in primates.

We may remark, however, that the most generally accepted explanation of such optic neuritis as being produced by two separate symmetrical foci of inflammation affecting the optic discs only, and occurring simultaneously with symptoms indicating a similar spinal lesion, is scarcely tenable in cases in which such optic neuritis is associated with local spinal tumours or injuries.

Conclusions.

(1) Optic neuritis of all degrees of severity may occur in connection with tumour, compression, myelitis or hæmorrhage affecting in some degree the upper part of the spinal cord.

(2) Headache and vomiting may be singly or together associated with neuritis from a local lesion of the cervical cord.

Appendix.

Case 1.—Mrs. M., aged 56, came under observation with the history that four years before she had begun to be troubled with difficulty in walking. This gradually became worse (although at times she could walk fairly well) until she became quite unable to use the lower limbs. To this, weakness of the arms was gradually superadded, and when she was first seen there was complete paralysis of all four limbs. There was also considerable although not complete impairment of the head movements. There was impairment of sensibility and the patient complained of a burning pain in the arms. There was also difficulty in passing water. One of us (J. T.) saw the patient at the suggestion of Sir W. R. Gowers, Mr. Victor Horsley and Mr. Marcus Gunn also saw her, and all four confirmed, besides the above symptoms, the presence of well-marked optic neuritis in both eyes.

The patient returned to her home, and about two months later died. The autopsy was performed about twenty-four hours after death by one of us (J. T.), kindly assisted by Dr. Henry Dobie and Dr. Herbert Dobie, of Chester. The spinal cord was examined and at the level of the third cervical root a small tumour about the size of a sparrow's egg was found outside the cord pressing on it. The most careful examination by all those who were present, of the brain and adjoining parts failed to reveal any intra-cranial growth. If any growth had been present it could scarcely have failed to be discovered.

Case 2.—H. J. T., aged 34 years, was admitted into the National Hospital under the care of Dr. Ferrier on April 14th, 1898, complaining of weakness of the legs and arms. The family history was good. He had acquired syphilis seven years before the onset of his illness.

His history was as follows: Five years before admission when in his usual good health he was suddenly seized with a fit in which he lost consciousness and was generally convulsed. A second similar fit occurred twenty minutes later, and a third attack in which the left side only was convulsed forty-five minutes later. After these attacks he is said to have had temporary weakness of the left arm and leg. He recovered perfectly in five months' time. A year later the fits re-appeared and have occurred since at irregular intervals. They were always preceded by several days of intense headache. Five months before admission he gradually became unable to stand,

subsequently the hands were slowly affected and he became unable to write.

On admission he was a well developed man. Mental state fair, memory poor. He could only hear a watch on contact on either ear.

Taste and smell were normal, vision good with each eye. No contraction of visual fields.

There was well marked double optic neuritis in each eye. The edges of the discs were quite invisible. Swelling = $6\frac{1}{2}$ D. left eye, and $4\frac{1}{2}$ D. right eye. There were some particles of effusion and small hæmorrhages.

The cranial nerves were normal.

There was absolute anæsthesia from the upper limit of the fifth cervical segment downwards, except over the soles of the feet and lower part of anterior tibial region, where all forms of sensibility were much impaired. There was complete loss of sense of position in all four extremities.

There was absolute flaccid paralysis of both upper extremities, but the upper part of both trapezii, the sterno-mastoids, and the diaphragm acted normally, and he could shrug his shoulders. The intercostals were completely paralysed. Marked wasting of the deltoids and spinati, severe wasting of the other muscles of the upper extremities with loss of faradic excitability were present, and absolute paralysis of both lower extremities, frequent reflex flexor spasms, complete reflex incontinence. The knee-jerks were exaggerated. There was double foot clonus.

He remained seven months in hospital and gained some power in both arms and legs. The faradic excitability returned in those muscles from which it had been absent. He had little or no pain. One attack of general convulsions occurred while he was in hospital. When discharged on November 3, 1898, the optic discs presented well-marked signs of post-neuritic atrophy.

Case 3.—Thomas L., aged 48 years, was admitted into the National Hospital, under the care of Dr. Hughlings Jackson, on February 28, 1896, complaining of pain in the neck and weakness in the arms and legs. He had had syphilis twenty-eight years before admission. The pain dated from a fall on to the head from a height of twenty-six feet, three years before admission. Six months before admission the pain became much more intense, and his limbs gradually became weak. When admitted there was marked spastic paralyses of both legs, the feeblest movements alone being possible. Left worse than right, com-

plete paralysis of left upper limb. All movements of right upper limb very feebly performed. There was complete analgesia and thermoanæsthesia to the upper limit of the fifth cervical root area. Sensation to touch was only very slightly impaired. There was incontinence of urine. The deep reflexes were much exaggerated. The optic discs presented no measurable swelling; the edges were nowhere distinguishable. There was some central exudation and exudation along the vessels. Stricture was present on the outer parts of both discs. The pupils were unequal, but reacted normally to light and to cocain. The other cranial nerves were normal.

Case 4.—E. B., aged 14 years, was admitted into the National Hospital, under the care of Dr. Buzzard, complaining of difficulty in walking and pain in the upper part of the back, radiating round the chest. There was a family history of tubercular disease. The history of the illness was as follows: Six months before admission she noticed a pain in the upper part of the back which ran round the top of the chest, and a fortnight later the knees began to feel numb, and the legs to become stiff in walking.

Three months before admission, on waking one morning, she found that she could not move either leg and had no feeling in them. She had not improved since this time. She had never had headache or vomiting during her illness.

On admission she was a well nourished, healthy looking girl. There was a marked angular curvature involving the third, fourth and fifth dorsal spines. The prominence was tender on pressure and there was pain in any movement of the spine. The special senses, cranial nerves and upper extremities were quite normal.

There was complete spastic paralysis of the lower extremities.

Absolute anæsthesia to all forms of sensibility below Poupart's ligament and the iliac crest. All forms of sensibility much impaired as high as level of the umbilicus. Slight blunting of sensibility as high as an inch above the nipples.

The optic discs were examined by Mr. Brudenell Carter, who found double optic neuritis more marked in the left than in the right eye.

On August 3 the neuritis was found to be much increased by Mr. Marcus Gunn. She had complained several times of frontal headache.

On September 23 the optic neuritis had almost disappeared. Voluntary power was returning in the legs, and sensibility had

been completely regained. On March 5, 1889, she left the hospital completely recovered except that she occasionally suffered with slight headache. The discs showed marked signs of past neuritis.

She was treated with rest and extension to the spine. Counter-irritation was applied to the back by means of the actual cautery. Cod-liver oil and iodide of iron were the drugs employed.

Case 5.—W. B., a gilder, aged 28 years, was admitted into the National Hospital, under the care of Dr. Buzzard, on September 27, 1892, having four months before fallen off his van into the road, striking the back of his right shoulder. He was under the influence of alcohol at the time, and was able to get up and walk home, a distance of 300 yards. He noticed nothing wrong till next morning, when, on waking, he was unable to move the right arm on account of severe pain. A fortnight later the pain had become more severe and both arms became powerless. During the next two months he slowly lost control of the legs and sphincters.

When admitted he was a well nourished, intelligent man. A watch was heard only on contact with left ear, the membrana tympani having been destroyed by otitis media.

Vision: right eye $\frac{6}{6}$ and J. 4; left eye $\frac{6}{24}$ and J. 14. The right optic disc was normal and there were about three dioptres of general hypermetropia. The left optic disc was much injected, outline concealed, veins large and distended.

The right pupil was persistently larger than the left, and both reacted normally, otherwise the cranial nerves were normal.

Motor system.—He was unable to move or turn in bed. In the lower extremities slight power of extension of knee and ankle were present. There was marked clasp-knife rigidity, and painful recurring reflex flexor spasm. The abdominal walls were rigid.

In the right upper extremity he had lost all power of flexing or extending the fingers and wrist. The biceps, deltoid and supinators and pronators of the forearms acted well. The triceps was a little weak.

In the left hand all intrinsic movements of the hand and fingers were lost, but the movements of the wrist were good. The triceps was slightly weak and the other muscles acted normally.

The intrinsic hand-muscles on each side were wasted. There was no change in the electric excitability either to faradism or galvanism. The right arm as a whole was smaller than the left.

Sensibility.—He complained of pain, persistent and increased on movement, in the region of the sixth and seventh cervical spines. These spines were tender to deep pressure. Sensation to touch was practically perfect all over the body. Sensibility to pain was lost or greatly impaired everywhere below the eighth cervical root area. Sensibility to cold was normal, that to heat eing slightly impaired over the affected area. There were some trophic changes on the skin of each hand, and profuse perspiration over both palms. The knee jerks were much exaggerated and there was double foot clonus. He was unable voluntarily to start the acts of micturition and defæcation. There was no spinal deformity.

On October 24 Mr. Horsley performed laminectomy opposite the sixth cervical vertebra, the leptomeninges were adherent to the theca, and a transverse ridge of bone projected backwards from the body of the vertebra and pressed upon the cord.

On October 26 the left optic disc was blurred and its outline was invisible. Surface seen with + 5 D. The right disc was injected and its edge was deficient in sharpness.

On November 3 there was marked improvement in movement in the arms and legs. He had had no pain since the operation. He was able to walk on January 29, and was completely recovered on September 19. The right optic disc at this time was normal; the left disc showed partial atrophy and white radiating striæ; its edge being invisible. There was no swelling. He has remained well since.

Case 6.—S. S., an engineer, aged 24 years, was admitted into the National Hospital, under the care of Dr. Ferrier, on July 7, 1899, complaining of loss of power and of feeling below the waist and incontinence of urine. Family history unimportant. Four years previously he had venereal disease with a non-indurated sore, and two years later he had an ulcerated throat, which rapidly healed under the influence of black wash.

In March, 1899, after having felt a curious cold feeling round his loins and some slight weakness of the legs for a week, he awoke one morning to find that he could not move either leg, and he was numb from the waist down. The sphincters were incontinent.

Six weeks later he was admitted into King's College Hospital under Dr. Ferrier. The knee jerks were noticed to be absent and the paralysis complete. A month later the right knee-jerk returned. The optic discs were normal at this time.

On admission there were no symptoms of intracranial disease. The mental state, special senses, and cranial nerves were normal.

Both optic discs were very red and their edges were blurred. There was radial white striation all over the discs. The swelling = 2 D. The arteries were pressing very much upon the veins.

The pupils were equal and re-acted normally.

There was absolute flaccid palsy of both legs, but the electrical excitability of the muscles was normal. The rectus abdominis did not contract below a point two inches above the umbilicus in the attempt to sit up (seventh dorsal). There was greatly diminished sensibility to touch below the seventh dorsal root distribution and almost complete analgesia, thermanæsthesia, and loss of sense of position below that area. There was absolute anæsthesia, incontinence of urine and fæces, and a deep sacral bedsore.

The plantar reflexes were extensor responses. The knee jerks could with difficulty be obtained with double reinforcement. The wrist and elbow jerks were normal.

On July 29 he complained of pain down both arms, and the grasps were found to be very weak. The arms were generally paretic, but the scapula-thoracic and humero-thoracic muscles were normal, as was also the deltoid.

Relative anæsthesia and analgesia were at this time present from the nipple downward, and there was some hyperæsthesia over the inner aspects of both upper limbs.

Three days later there was complete anæsthesia and analgesia to a line round the thorax 1½ inches above the nipples.

On November 4 the upper extremities had slowly and completely recovered power. The anæsthesia was still almost complete below the nipples. The swelling of the optic discs had subsided, their edges were woolly and indistinct. There were obvious signs of old exudation on the surface of both discs.

Case 7.—Samuel C., aged 36 years, was admitted into the National Hospital under the care of Dr. Ferrier on May 27, 1896, complaining of paralyses of the legs, headache and dysuresis. He denied venereal disease. He had malaria in America seven years before admission. Nine months before admission he was seized with dull pain in the back and numbness in the legs as he was walking. Weakness in the legs and difficulty in passing water followed a few hours later, and at the end of forty-eight hours the legs were completely paralysed, and he was numb up to his waist.

Three weeks later he began to recover sensation in the legs. At the time of onset of his illness he had much headache and general malaise. When admitted there was complete spastic paraplegia, and sensibility to all forms was diminished below the fourth dorsal root level. The deep reflexes were much exaggerated and there was double foot clonus. The pupils reacted normally, and the cranial nerves, other than the optic, presented no peculiarity.

Optic Discs.—Right, edge indistinct and woolly; veins distended, some connective tissue along vessels; some central filling and many fine vessels over disc; no swelling. Left similar, but less markedly abnormal.

Case 8.—W. J. E., a butcher, aged 34 years, was admitted into the National Hospital under the care of Dr. Bastian on August 23, 1892, complaining of loss of power and sensibility in the legs and incontinence. His health, previous to this illness, had been good. He had had five attacks of gonorrhœa. Five years before admission he had symptoms which were presumably those of syphilitic infection. In March, 1892, he was laid up with an illness accompanied by pain in the back and shivering. He was acutely ill for a fortnight, at the end of which time he noticed that he was losing power in the left leg. In three weeks he completely lost power in both legs, and in the lower part of the trunk.

On admission he was completely paralysed below the fifth intercostal space, the diaphragm, which acted well, being excepted. The lower extremities were rigid, and there were at times reflex flexor spasms. The upper extremities were normal. There was a band of extreme hyperæsthesia extending round the thorax, its upper limit being the third intercostal space in front and its lower limit the lower edge of the fifth rib.

There was complete loss of tactual sensibility over the fourth and fifth lumbar and all the sacral areas of the left side, and partial loss over the same areas of the right side. There was analgesia over the right half of the body and right leg, limited sharply above by the sixth rib in front and eighth dorsal spine behind and by the mid-line of the body.

Thermo-anæsthesia extended as high as the third intercostal space. There was complete loss of sense of position and of passive movement in the lower extremities.

Knee-jerks exaggerated. Double foot clonus.

Complete loss of control of sphincters.

Pupils small and equal and re-acted to light and during accommodation.

Both optic discs full coloured and edges decidedly blurred though still traceable, the retinal veins were over full, probably an early stage of papillitis. He suffered occasionally with headache and vomiting.

On December 6, 1892, the optic discs were normal.

Case 9.—G. M., aged 22 years, was admitted into the National Hospital, under the care of Dr. Bastian, in February, 1899, suffering with complete loss of power in the legs and incontinence of sphincters.

When 6 weeks old she developed signs of congenital syphilis. When 7 years old she suffered with interstitial keratitis and her sight became in consequence somewhat impaired. When 13 years of age some lateral curvature of the spine was noticed. At 14 she had chorea. When 20 years old in the course of a week she lost the feeling and use in her legs and became completely incontinent. Some weeks after her sight began to fail and her memory became much impaired. She had not improved up to the time of her admission. When admitted she could perceive light with the left eye and could only count fingers with the right. Both fundi shewed advanced post-neuritic atrophy. Leucomata were present in both cornea. Old iritic adhesions were present in each eye and the pupils did not respond to light, the right alone contracting slightly on convergence. The other cranial nerves were normal.

There was absolute spastic paraplegia below the chest, and complete anæsthesia to all forms below the ninth dorsal level. Marked impairment to pain and touch was present up to and including the second dorsal root area.

Case 10.—A. S., a schoolgirl, aged 15 years, was admitted into the National Hospital, under the care of Dr. Hughlings Jackson, on October 5, 1898. The family history was good and she had always been healthy. A fortnight before the onset of this illness she fell off a stile and hurt her back slightly. Fifteen months before admission and a fortnight after the fall on her back, on getting into bed one night she felt a sudden sharp pain between the shoulders and "went out stiff" in her arms and legs. She completely lost power in the arms and legs at once and could not feel below the upper part of the chest; there was complete loss of control over the sphincters.

Up to the time of her admission the arms improved somewhat,

but the legs remained completely powerless. Flexor spasm and bed sores troubled her greatly.

On admission there was slight analgesia over the whole body below the fourth cervical area.

Great atrophy of all intrinsic hand muscles and of ulnar flexors. Other muscles of the upper extremity normal. Double "main en griffe."

Intercostals completely paralysed. Diaphragm acted well. Absolute spastic paraplegia in trunk and legs. Double foot clonus.

Mr. Gunn examined the eyes and made the following note:—Well marked optic neuritis in either eye. Swellings = 3 D. Edges of discs quite blurred. Several small hæmorrhages near the disc in both eyes. Pupils equal and normal, but the left did not re-act to cocain and the right only re-acted slightly.

Case 11.—*Post-neuritic optic atrophy in chronic "myelitis," upper limit of lesion in the fifth cervical segment.*—J. C., aged 41 years, a police constable, was admitted into the National Hospital under the care of Dr. Bastian on June 25, 1895, complaining of weakness of the legs. There was a history of venereal disease (nature uncertain), nineteen years before admission. He had noticed gradually increasing weakness in the legs for fifteen years and had been unable to work for seven years. When admitted his condition was one of marked spastic paraplegia involving both arms and legs. He could still walk with a stick. The pupils reacted normally and there was abnormality in the cranial nerves other than the optic. Vision R.E. $\frac{6}{36}$, L.E. $\frac{6}{24}$, J. 4 in both eyes.

Both discs presented a blurred, woolly, indistinct margin, with some connective tissue along the vessels, which were very small. Both discs were very pale, and there was evidence of old exudation in the centre of each.

The upper extremities were weak and rigid. There was slight anæsthesia to all forms below the fifth cervical area. Some rigidity was present in all four limbs. There was no local wasting of muscles. All the deep reflexes were exaggerated and there was double foot clonus.

The nature of the lesion in this case was uncertain. The upper limit of its extent in the spinal cord was definitely the fifth cervical segment. The condition of the optic discs was that typical of consecutive atrophy following a neuritis of moderate degree. This atrophy was first seen by Dr. James Taylor two

years before admission, and the condition of the discs and vision had not changed during the period he was under observation. He had never at any time suffered with headache, vomiting, or cerebral symptoms.

Case 12.—E. K., a compositor, aged 28 years, was admitted into the National Hospital under the care of Dr. Hughlings Jackson on June 18, 1900. His health had been very good before the onset of this illness. He had not had symptoms of lead poisoning and he denied venereal disease. At Easter, 1900, he suffered with lumbago for four days and at the end of the same week he noticed that his sight was failing and that his legs were numb and weak. He went to Moorfields and came under the care of Mr. Morton, who discovered that optic neuritis was present. He was admitted, and his sight gradually improved, but the legs became more and more weak and the numbness gradually involved the trunk from below up, and he developed dysuresis. He had never had headache during the illness, but had vomited several times during the first few weeks. A girdle sensation of tightness had been present above the waist for some weeks.

When admitted to Queen Square, vision was Rt. eye $\frac{6}{6}$ and J. 1; left eye $\frac{6}{9}$ and J. 14. Left field concentrically contracted. Both discs showed post-neuritic atrophy L > R.

The left pupil did not re-act to light: otherwise the cranial nerves were normal. Slight weakness and marked tremor on movement in the intrinsic muscles of both hands. Muscles of the abdominal wall rigid. The lower extremities were weak and spastic, the right much more so than the left. The knee jerks were exaggerated and ankle clonus was present. He was unable to walk.

There was anæsthesia to light touches everywhere below the level of the ensiform process. The deep reflexes were increased in the lower extremities, and the plantar reflexes were extensor in type. He had difficulty in starting the act of micturition. He left the hospital on August 29, able to walk well though still showing a spastic ataxic gait; the anæsthesia and dysuresis had disappeared.

TABLE OF 240 CASES OF LOCAL DISEASE OF THE SPINAL CORD, FROM THE RECORDS OF THE NATIONAL HOSPITAL, 1893—1899 INCLUSIVE.

Nine of the Writers' Cases are here included.

The figures in dark type refer to cases in which definite papillitis or post-neuritic atrophy has been observed; those in ordinary type to cases in which the optic discs presented no changes. All cases in which albuminuria, lead poisoning, or extensive vascular degeneration was present have been excluded. The spinal segment referred to denotes the approximate upper limit of the lesion.

In 86 cases either there was no record of the condition of the optic discs or the records were too scanty to determine even approximately the limits of the lesion.

The remaining 154 cases are here tabulated:—

	C.5.	C.6.	C.7.	C.8.	D.1.	D.2.	D.3.	D.4.	D.5.	D.6.	D.7.	D.8.	D.9.	D.10.	D.11.	D.12.	L.1.	L.2.	L.3.	L.4.	L.5.	
Myelitis	2, **2**	**1**	1, **1**	1, **2**	2, **1**	1	0	4	3	2, **1**	4	2	12	9	3	4	7	2	1	—	—	= 60, **8** = 68
New growth	1	1, **1**	1, **1**	2	1	**1**	0	0	0	0	2	1	1	0	1	0	2	2	1	0	1	= 17, **3** = 20
Hæmatomyelia .. .	1, **1**	0	**1**	0	1	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	= 4, **2** = 6
Fracture dislocation ..	0	0	**1**	1	**1**	—	—	—	—	1	—	—	1	1	—	1	2	1	1	—	—	= 9, **2** = 11
Pachymeningitis ..	**1**	—	—	—	**1**	—	—	1	—	2	—	—	—	—	—	—	—	—	—	—	—	= 3, **2** = 5
Spinal caries	**1**	0	1, **1**	2	4	1	2	4	4	4	4	3	3	3	4	1	1	1	—	—	—	= 42, **2** = 44
Optic discs normal ..	4	1	3	6	8	2	2	9	7	9	10	6	17	13	9	6	13	6	3	0	1	=135
Neuritis present.. ..	**5**	**2**	**5**	**2**	**3**	**1**	0	0	0	**1**	0	0	0	0	0	0	0	0	0	0	0	=**19**

	With optic neuritis.	Without.
Lesions affecting cervical enlargement	18	24
" " dorsal region	1	88
" " lumbo-sacral region	0	23

Table of Published Cases.

The numbers in the sixth column refer to the upper limit of the lesion as verified or deducible from the symptoms. In several cases the ophthalmic signs have been recorded in great detail, but the spinal signs with so little, that it has been impossible to state definitely the position of the lesion.

Author.	Reference.	Sex.	Age.	Lesion.	Situation.		
Abadie	*Bull. Soc. Chir.*, Jan. 12, 1876						
Achard and Guinon .	*Arch. Med. Exp.*, 1889, p. 696 ..	M.	30	Diffuse myelitis ..	Cervico-dorsal + at D. 3 ..	O. N. preceded	+
Allbutt	"Gower's Med. Opthal.," 3rd ed., p. 189	—	—	Myelitis	Upper dorsal		
Bielschowsky ..	*Neur. Centralbl.* 1901, p. 457 ..	M.	43	,, ...	C. 8—D. 1		
Bull	*Amer. J. Med. Sc.*, July, 1875 ..						
Chauvel	*Bull. Soc. Chir.*, 1880, p. 512 ..	M.	23	,, ...	D. 1		
Chisholm	*Arch. f. Ophth.*, xi., No. 2, June, 1882	M.	28	? Hæmatomyelia ..	D. 1—2	O. N. preceded	
Devic..	*Cong. Franç. de Med.*, 1894, p. 434..	F.	45	Myelitis	Upper dorsal..	.. .. ..	+
Drake-Brockman ..	*B. M. J.*, 1892, pt. 2, p. 77	M.	19	,, ...	Cervical	Vomiting	
Dreschfield	*Lancet*, 1882, p. 8052	F.	38	Softening	Cervical enlargement 1½ ins.	.. .. ..	+
	B. M. J., 1894, p. 474	M.	41	Myelitis	D. 1	.. .. ..	+
		M.	23	Disseminate myelitis	Throughout cord	.. .. ..	+
Elschnig	*Arch. f. Augenheilkunde*, xxvi. ..	M.	26	Myelitis	Mid-dorsal ?	Argyll Robertson pupils	
		M.	22	,, ...	?	,,	
		M.	38	,, ...	?	,,	
		F.	31	,, ...	?		
Erb and Steffan ..	*Arch. f. Psych.*, x., p. 146	M.	50	,, ...	Upper dorsal		
Eskridge	*J. N. and M. Dis.*, N.Y., 1890 ..	M.	49	,, ...	C. 8 and D. 1	.. .. ..	+
Firth	*Practitioner*, 1886	M.	23	Fracture dislocation	C. 6, 7. 8; D. 1		
Gault	Lyon, 1894						
Gowers	*Med. Ophthalmoscopy*						
Hoffmann	*Neur. Centralbl.*, 1896, S. 671 ..	M.	48	Myelitis	Cervical enlargement		
		F.	56	,, ...	Upper dorsal		
Katz	*Arch. für Ophth.*, xlii.	F.	56	,, ...	Cervical enlargement ..	.. .. ..	+
Knapp	"Tagebl. der 58 Versamml. deut. Naturf. und Aertze." Strasburg, 1885, p. 489	M.	32	,, ...	D. 1		
Mahokian	*Inaug. Dissert*, Berlin; reference with case, No. 24	M.	23	,, ...	C. 8—S. 4	.. .. ..	+
Mooren	*Ophth. Mitt.*, Berlin, Hirschwald						

Mooren	*Ophth. Mitt.*, Berlin, Hirschwald, 1894							
Nonne	*Arch. f. Psych.*, lxxxiii., f. 2, 1900	F.	15	Sarcoma	C. 2	.. .. ..	+
Noyes	*Arch. f. Ophth.*, ix., 1880, pt. 11, p. 129	M.	25	No details			
	Arch. f. Augenheilkunde, 1881						
Rieger and Forster..	*Arch. f. Ophth.*, xxvii, 3, p. 109 ..	—	—	Myelitis	No details		
Ripamotti	*Atti dell'assoc. Med. Lomb.*, Milan, 1896						
Rumpf	*Deut. Med. Woch.*, 1881, p. 442 ..	F.	32	,, ...	Details deficient		
Schatz	*Deut. Med. Woch.*, 1893, p. 615 ..	M.	19	,, ...	Lower cervical		
Schluster and Mendel	*Neur. centralbl.*, 1899, p. 1088 ..	M.	23	Diffuse myelitis ..	Complete destruction of cord below C. 3	.. .. ..	+
				,, ,, ..	C. 2, &c.	.. .. ..	+
				Dissem. ,, ..	Largest focus at D. 2 ..	.. .. ..	+
Schlüter, F.	"Ueber opt. neur.," *Marg. Dis.*, Berl., 1882	M.	49	Myelitis	?		
Sequin	*J. Neur. Ment. Dis.*, N.Y., April, 1880	M.	35	,, ...	Upper dorsal	.. .. ..	+
Sharkey	*Med. Times*, 1884, i., p. 812 ..	F.	19	Softening	Cervical enlargement ..	.. .. ..	+
Sharkey and Lawford	BRAIN, viii., 133 (Abstract) ..	F.	17	Myelitis	Cervical region, upper and lower	O. N. preceded	+
Taylor, F.	*Guy's Hosp. Reports*, 1896, 53, p. 45	F.	33	,, ...	Cervical		
Taylor, James ..	*Allchin's Medicine*, vol. iii., p. 394	—	—	Several cases under	personal observation, referred	to	
Taylor and Collier ..	BRAIN, Winter No., 1901	F.	56	Myxoma	C. 3	.. .. ..	+
		M.	34	Tumour	C. 5		
		M.	48	? Tumour	C. 5		
		F.	14	Spinal caries ..	Cervical		
		M.	28	Fracture Dislocation	C. 6		
		M.	24	Myelitis	Cervical		
		M.	36	,, ...	Upper dorsal		
		M.	34	,, ...	D. 2		
		F.	22	,, ...	D. 2		
		F.	15	? Hæmatomyelia ..	C. 4		
		M.	41	Chronic Lesion? ..	C. 5		
		M.	28	Myelitis	Upper dorsal		
Thorowgood	*Clin. Trans.*, viii., 1875, p. 80 ..	—	—	Trauma to neck ..	Cervical		
Uhthoff	*Berl. Klin. Woch.*, 1885, No. 16 ..	—	—	Three cases of "multiple sclerosis" with optic neuritis			

A CONTRIBUTION TO THE CLINICAL SIGNIFICANCE OF ABSENCE OF THE TENDO-ACHILLIS-JERK.

BY EDWIN BRAMWELL, M.B., M.R.C.P.LOND. AND EDIN.

CONTENTS.

(1) Introductory remarks.
(2) Method of examining the Achillis-jerk.
(3) The Achillis-jerk in health and disease; an investigation based upon the examination of 1,009 individuals.
(4) Conclusions.

INTRODUCTORY REMARKS.

THE following investigation was undertaken in the hope of ascertaining more exact knowledge as to the diagnostic significance of absence of the Achillis-jerk.

It is only within the last two or three years that any diagnostic value has been attached to this jerk, and that probably for two reasons. Firstly, since in the majority of cases in which the Achillis-jerk is altered, the knee-jerk is similarly affected, the examination of the Achillis-jerk consequently affords little or no additional diagnostic data; while, secondly, the idea is commonly prevalent that the Achillis-jerk is absent or not obtainable in a certain proportion of healthy individuals.

The first question which it is proposed to consider in this paper is the state of the Achillis-jerk in "health"; in other words, is absence of the Achillis-jerk always to be regarded as pathological? So long as doubt exists upon this point, it is obvious that absence of the Achillis-jerk will never be regarded as a physical sign of diagnostic importance.

In the latter part of the paper, the results of the examination of a number of patients suffering from various forms of disease are recorded, and conclusions drawn as to the diagnostic value of absence of the jerk.

Before referring to the state of the Achillis-jerk in health and disease, it is, however, advisable to describe the methods best adapted for examining the jerk. This is all the more necessary since it appears probable that defective technique in examining the jerk is largely accountable for the little attention which it has hitherto received, as well as for the uncertainty which exists regarding its clinical significance.

Method of Examining the Tendo-Achillis-Jerk.

The kneeling position, as recommended by Babinski, is without doubt that in which the Achillis-jerk is most conveniently examined and easily obtained.

The patient kneels on a chair with his feet projecting a few inches over the edge. In this position the muscles of both the front and back of the leg are usually completely relaxed. A hammer of some kind is necessary with which to strike the tendon. A wooden stethoscope with a rubber ring attached around the ear-piece serves very well for this purpose. If no hammer is at hand the edge of a thin book may be used.

The tendon is struck *sharply*, close to its insertion into the os calcis, and, if the jerk is present, the stroke is immediately followed by a quick plantar flexion-movement of the foot at the ankle joint.

The true jerk is a quick movement occurring immediately *after* the stroke, and is quite distinct from the slight movement of the foot which occurs as a mechanical effect in cases in which the reflex is lost.

If the jerk is not obtained, the observer must make sure that the muscles of the legs are not contracted. It is especially important in comparing the jerks on opposite sides of the body, to see that the leg muscles on both sides are fully and equally relaxed.

The various methods of reinforcement recommended by Jendrassik,[1] such as pulling on the hands, &c., should be adopted where the jerk is not obtained. A succession of sharp strokes sometimes elicits the reflex when a single stroke fails to do so. In one case in which the Achillis-jerk was difficult to get, Dr. Byrom Bramwell found that striking the calf muscle several times with the hammer appeared to reinforce the jerk. The Achillis-jerk, when present, can usually be readily obtained with boots on, in the kneeling position.

When the patient is unable to kneel, the examination must be made in the prone position.

If the jerk is well marked, it is easily obtained in this position. This method of examination is, however, much more difficult than the kneeling position, and its successful application requires considerable experience. The following is the procedure which the writer adopts, when it is necessary to examine the patient in the prone position.

The patient is asked to turn from his back slightly towards the side of the limb about to be examined. The limb is flexed at the knee almost to a right angle and is then laid on its outer side upon the bed. In this way, complete muscular relaxation is usually secured. The ball of the great toe is grasped lightly between the finger and thumb of the left hand and the foot is dorsi-flexed sufficiently to put the tendo Achillis slightly on the stretch. The inner edge of the tendon is then struck with the hammer, and the slightest plantar movement of the foot is felt by the hand which grasps the patient's foot. Should no jerk be obtained, the tension on the tendon should be slightly increased and the jerk again tested. Reinforcement should then, if necessary, be employed.

The State of the Tendo-Achillis-Jerk in Health and Disease.

The following investigation is based upon an examination of 1,009 persons from various communities :—

[1] *Neurolog. Centralblatt*, 1885, S. 412.

Fifty Children from the Medical Wards of a Children's Hospital.

Through the kindness of Drs. Burn Murdoch, Melville Dunlop and John Thomson the writer had the opportunity of examining the Achillis-jerks in 50 patients in the Royal Hospital for Sick Children, Edinburgh; none of them, so far as is known, were suffering from any form of nervous disease.

Almost all of these cases were examined in bed in the prone position, and in every case, with one exception, both Achillis-jerks were at once obtained. The inability to obtain the jerks in the single exceptional case was due to the fact that the child, who had recently been admitted, was fretful and would not relax its leg muscles. On a subsequent occasion, both jerks were found to be quite active in this case.

Two Hundred and Nineteen School Children, Eight to Fifteen Years of Age, including 111 *Deaf-mutes.*

Dr. Lockhart Gillespie and the House-Governor of Donaldson's Hospital, Edinburgh, kindly gave the writer permission to examine the inmates of that Institution.

The Achillis-jerks were examined in 219 children, varying in age from eight to fifteen years. Of these children, 111 (60 boys and 51 girls) were deaf-mutes, while the remaining 108 (67 boys and 41 girls) were healthy school children. All were examined in the kneeling position.

Both Achillis-jerks were readily obtained in every case with four exceptions; all of whom were boys and all deaf-mutes. In none of these four cases in which the Achillis-jerks were both absent was there rigidity of the leg muscles. A day or two later, the four exceptional cases were again examined, and in one the jerks were obtained, though with difficulty. In the remaining three the absence of the Achillis-jerks was confirmed. In one of these cases, the

knee-jerks were absent, in another they were very sluggish, while in a third they were quite active. In none of these cases was any cause discovered to account for the absent Achillis-jerks.

One Hundred Convalescent Fever Cases.

The writer's thanks are due to Dr. Claude B. Ker, who kindly gave him the opportunity of examining 100 convalescent patients in the City Fever Hospital, Edinburgh, the great majority being children and young adults.

The patients were convalescing from the following fevers:—scarlet fever (69 cases), enteric (14), diphtheria (10), erysipelas (5), and quinsy (2).

In all the cases, with four exceptions, both the knee and Achillis-jerks were present. In the case of two patients who were suffering from post-diphtheritic paralysis, both the knee and Achillis-jerks were absent. In one instance (a convalescent from scarlet fever), the left Achillis-jerk was not obtained, but two days later was found to be well marked. In the last case, also post-scarlatinal, neither Achillis-jerk could be obtained although the knee-jerks were brisk. The inability to obtain the jerks in this case was ascribed at the time to rigidity of the leg muscles, but, unfortunately, the writer had not a further opportunity of examining the case as the patient left the hospital on the following day.

Eighty Medical Students.

Eighty medical students were examined in the kneeling position with boots or shoes on; in every case but one both jerks were well marked. In this instance, they were obtained after the student had removed his boots; reinforcement, however, had to be resorted to before one of the jerks became evident. The knee-jerks were somewhat sluggish in this case.

Two Hundred and Thirty Individuals Over 40 Years of Age.

Through the kindness of Dr. Edward Carmichael and Dr. R. S. Aitchison, and the Governors of the Craiglockhart and Craigleith Poorhouses, Edinburgh, opportunity was afforded of examining 230 inmates of these institutions, including 157 men and 73 women.

The great majority of the individuals examined were well past middle age. *The jerk was tested in all cases in the kneeling position, the patients wearing either light shoes or stockings only.* Reinforcement was used in all cases where the jerks were difficult to obtain. The results in relation to age are interesting and are well shown in the following table:—

The Achillis-jerk in Persons over 40 Years of Age.

Analysis of 230 Cases.

Age.	No. of Cases.	Cases in which Achillis-jerks not obtained.			Percentage of Cases in which Achillis-jerks affected at different ages.
		One.	Both.	Total.	
Over 80 years	15	2	10	12	80 per cent.
70-80 years	65	5	36	41	63 ,,
60-70 ,, ...	80	3	25	28	35 ,,
50-60 ,, ...	42	1	4	5	12 ,,
40-50 ,, ...	28	—	—	—	—

It is probable that if these cases had been again examined, in some the Achillis-jerks would have been obtained; the percentages in the last column are for this reason almost certainly too high. Admitting this, however, a justifiable conclusion to be deduced from this investigation is, that in persons past middle age, the Achillis-jerk is not always obtainable, and further, with increasing years the proportion of cases in which the Achillis-jerk is absent becomes increasingly greater.

In old people, the Achillis-jerk when obtained is usually much less active than in young or middle aged adults. In old people in whom the Achillis-jerks are well marked, the individuals are often unusually active and frequently appear to be endowed with a vitality beyond their years.

One Hundred Insane Patients.

Through the kindness of Dr. Clouston, the writer had the opportunity of examining the Achillis-jerks in 100 male patients in the Royal Asylum, Morningside, Edinburgh. In these cases, both Achillis-jerks were present in eighty-nine cases, while one or both were absent in eleven cases. Of the eleven cases in which the Achillis-jerks were absent, five were cases of general paralysis, five were cases of senile dementia, and one was a case of alcoholic insanity.

In three of the cases of general paralysis, both the Achillis-jerks and knee-jerks were absent. In one case, the right, in another the left, Achillis-jerk was absent, the knee-jerks and opposite Achillis-jerk being present. In one case, the right knee-jerk was absent, the left knee-jerk and both Achillis-jerks being present.

In all of the five cases of senile dementia included in the eleven exceptional cases, both knee-jerks were present; while in four, both Achillis-jerks were absent. In the remaining case, the left Achillis-jerk was present. All of these patients were well advanced in years, four being from 63 to 66 years of age. The fifth patient stated that he was 55, but his appearance was that of a very much older man.

In the case of alcoholic insanity both Achillis-jerks were lost, the knee-jerks being unaffected.

One Hundred Patients from the Medical Wards of a General Hospital.

For kind permission to examine 100 hospital patients in the Edinburgh Royal Infirmary, suffering from a variety of medical diseases, the writer's acknowledgments are due to Professor Wyllie, Drs. Byrom Bramwell, Alexander Bruce and Alexander James.

The patients examined were suffering from the following conditions:—

	No. of cases.	One or both Achillis-jerks absent or greatly diminished.
Cardiac disease of various forms	16	1
Rheumatism	14	1
Dyspepsia, including gastric ulcer	13	2
Pneumonia	11	0
Diabetes Mellitus	7	2
Phthisis	7	0
Chlorosis	6	0
Aortic aneurism	4	2
Acute Bright's disease	3	0
Exophthalmic goitre	3	0
Pernicious anæmia	2	0
Diabetes insipidus	2	0
Cancer of liver	2	0
Pericarditis, psoriasis, lupus, leucocythæmia, cancer of stomach, arrested bodily development, chronic bronchitis, gout,* catarrhal jaundice, and nocturnal eneuresis—of each 1 case	10	1*
Total	100	9

Both Achillis-jerks were present and well marked in all but nine cases.

The following is a brief record of the exceptional cases:—

Case 1.—J. J., labourer, aged 37 (under the care of Dr. B. B.), suffering from diabetes mellitus. Both Achillis- and knee-jerks were absent.

Case 2.—J. B., iron-planer, aged 34 (under the care of Dr. B. B.), suffering from diabetes mellitus. Both Achillis-jerks and knee-jerks were absent.

It is a well known fact that the deep reflexes are often absent in diabetes mellitus. In one case examined by the writer, the knee-jerk was absent and the Achillis-jerk present (the patient had only one leg). Babinski [1] has examined a diabetic in whom the knee-jerks were normal, while the Achillis-jerks were lost.

Case 3.—R. S., joiner, aged 34½ (under the care of Dr. B. B.), complained of dyspepsia from which he had suffered for seventeen years, he had been worse since a severe attack of influenza ten months previously. Both the knee- and Achillis-jerks were absent.

He was a temperate man as regards alcohol, and absolutely denied syphilis and gonorrhœa; there was no history of secondary syphilitic symptoms. He had been married ten years; his wife had had a miscarriage at six weeks, a few months after marriage, which was said to have been caused by a fall. There was one child alive which was said to be perfectly healthy: there had been two miscarriages at a later date, attributed by a gynæcologist to some uterine trouble. There were no signs of tabes or G. P. I. The pupils were equal, regular and reacted well to light. There was no ataxia or Rombergism and no thoracic or ulnar analgesia. No history of shooting pains or bladder trouble. His mental state appeared perfectly satisfactory. There was no weakness of the legs or tenderness of the calves and no history of diphtheria. No sugar in the urine.

Possibly the loss of the knee- and Achillis-jerks may have been the result of the attack of influenza above referred to (?).

Case 4.—J. C., labourer, aged 48, was admitted into the R. I. E. (under the care of Dr. B. B.), suffering from a typical gouty swelling of the left hand which rapidly disappeared under appropriate anti-gouty remedies.

In this case both Achillis-jerks were absent while the knee-jerks were present although not well marked. There was no history of syphilis.

On enquiry, it was ascertained that *the patient had had two separate attacks of sciatica* between two and three years previous to his admission to hospital, affecting first one leg and then the other.

[1] *Revue Neurologique*, 1901, p. 482.

The occurrence of absence of the Achillis-jerk in cases of sciatica will be referred to later (see p. 570).

Case 5.—H. F., labourer, single, aged 38 (under the care of Dr. B. B.), was admitted to the R. I. E. on account of dyspeptic symptoms.

The patient complained of shooting pains in both legs below the knee.

On examination it was found that the knee-jerks were active and equal but that the Achillis-jerks were both absent.

The calf muscles were exceedingly tender, but there was no perceptible weakness of the extensors of the feet. The patient stated that he had been drinking heavily for some months previously, and he himself attributed his symptoms (dyspepsia, &c.), to this cause.

The patient admitted having had gonorrhœa, but denied syphilis, and there was no history of secondary symptoms. A thorough examination failed to elicit any signs suggesting tabes—the pupils reacted actively to light, there was no thoracic or ulnar analgesia, no affection of the bladder or rectum, and no derangement of the sexual functions.

A diagnosis of *early peripheral* (*alcoholic*) *neuritis* seemed perfectly justifiable on these facts.

The condition of the Achillis-jerk in peripheral neuritis is referred to on p. 569.

Case 6.—A. F., domestic servant, aged 25. This patient was recently admitted to the R. I. E. (under the care of Dr. B. B.) supposed to be suffering from subacute rheumatism. She complained of pains in her legs and back. The history of her illness was as follows:—Six weeks previously she first noticed a sharp pain behind the right knee which afterwards extended up and down the leg, and in the course of a few days, spread to the opposite leg. At the same time she had a severe pain in the lower part of the back. As the pains did not disappear with anti-rheumatic remedies, she was recommended for admission to the hospital. There had been no swelling nor pain in any of her joints. She had not been feverish, nor had she sweated. She had never suffered from rheumatism nor was there any history of injury to the back. There was no family history of rheumatism or tubercle. On examination she was a well nourished, healthy-looking girl. Her temperature was normal, her tongue clean and skin dry. There was no swelling of any of the joints. The pain which she complained of was situated over the upper

sacral region and was, she said, made worse by movement. The pains in the legs, which were not so severe as they had been, affected both legs equally, and were felt down the back of the legs, especially behind the knees. The pains were shooting in character. There was slight tenderness on pressure over the sciatic nerves. The power of the muscles of both the front and back of the leg was markedly diminished, as was that of the hamstrings on both sides, while no distinct weakness could be made out in the extensors of the knee, the muscles of the abdomen, or upper limbs.

A very careful examination failed to reveal the slightest evidence of impairment or hyperacuity of sensation either to touch or pain over the lumbar or sacral root areas. There was no tenderness of the leg muscles. The knee-jerks were somewhat increased as was the left Achillis-jerk, while the *right Achillis-jerk was absent*. There was a double extensor response, best marked on the left side. There had been no disturbance of the bladder reflex, no delay in commencing the act, no incontinence, no precipitate micturition. Some weeks previous to admission, she had for a few days experienced a burning sensation when passing water. The patient stated that she had no vaginal discharge. The rectal functions were unaffected. There was no irregularity of the spine and no tenderness on pressure. The functions of the special senses, cranial nerves and upper extremities were perfectly normal.

In this case the weakness of the hamstring and leg muscles, together with the pains in the back and legs, and the double extensor response, pointed to a *local lesion involving the last lumbar (?) and upper sacral roots*, or that part of the spinal cord with which they are directly connected. What the exact nature of this lesion was it was difficult to say. *In this case the absent Achillis-jerk on the right side was of undoubted localising value.*

Case 7.—J. D., mason, aged 53 (under the care of Dr. B. B.), admitted to the R. I. E. with definite signs of a thoracic aneurism. He had never been a heavy drinker.

The right Achillis-jerk was present though difficult to elicit, the left Achillis-jerk on the first occasion on which it was tested could not be obtained, but subsequently, when the patient was examined in the kneeling position, a very slight movement of the foot was once or twice seen.

The knee-jerks were increased and equal.

There was a double extensor response.

The right pupil was slightly larger than the left and oval in

shape, the result of an old iritis. *Both pupils reacted very slightly to light, rather better on accommodation.*

The patient admitted having had gonorrhœa thirty years previously, but denied syphilis, and there was no history of secondary symptoms. No history of sciatica or shooting pains in the legs; no thoracic or ulnar analgesia. Some dribbling of urine lately, but this may have been accounted for by an enlarged prostate.

The patient died soon after leaving the hospital. At the autopsy two large aneurisms were found, one of them, situated opposite the bodies of the ninth, tenth and eleventh vertebræ, had eroded these considerably. The arteries were extremely atheromatous.

Case 8.—A., aged 57 (under the care of Professor Wyllie), suffering from aortic incompetence.

Both Achillis-jerks were absent (examined on two occasions, both in the kneeling and prone positions), while the knee-jerks were present and about normal in activity.

The patient denied syphilis, and there was no clear history of secondary symptoms. *His pupils, however, were slightly irregular and scarcely reacted at all to light, though well on accommodation.* There were no other signs suggestive of tabes or G. P. I.; no evidence of peripheral neuritis, and no history of sciatica. His wife had had thirteen children, *six of whom died in infancy.*

Case 9.—J. R., labourer, aged 34 (under the care of Dr. B. B), suffering from a thoracic aneurism. Both Achillis-jerks were absent. The activity of the knee-jerks was below the normal, the right being less active than the left. The pupils measured 2½ mm., were equal and regular and reacted to light, although the contractions were not very brisk. They contracted actively on accommodation. There were no other signs suggesting tabes and nothing pointing to peripheral neuritis. He had not had sciatica. The patient was married, but had had no children. He denied syphilis. Unfortunately, he left the hospital before additional data bearing upon the question of previous syphilis had been enquired into.

Although in none of the three cases last described is there absolutely certain evidence of previous syphilis, the writer suggests syphilis as the probable cause of the loss of the Achillis-jerks in these cases.[1] In other words, he

[1] In connection with these cases it is interesting to note that Charles L. Greene, of St. Paul, in his work on "Examination for Life Insurance," referring to the knee-jerk, remarks: "The author has several times found it to be lacking in aneurism of the aorta." Rebman Limited, London, 1901.

believes that loss of the Achillis-jerk may occur as a result of syphilis unaccompanied by other signs of tabetic degeneration, just as the Argyll Robertson pupil is sometimes observed as a sequel of syphilis in the absence of other signs of tabes, G. P. I., &c.

The data upon which the writer bases this statement are as follows:—

(1) It is very exceptional to meet with absence of the Achillis-jerk, where a satisfactory explanation cannot be ascertained to account for it.

(2) The three cases mentioned above were all suffering from aortic disease (two from aortic aneurism), a fact which suggests some common etiological factor.

(3) Syphilis is an important factor in the production of aneurism and aortic disease.

(4) Although in none of the three cases above mentioned is there absolute evidence of syphilis, the aneurismal or aortic lesion in itself makes it not improbable that the patients had at some previous time suffered from syphilis. Further, in two of the three cases, apart from the aortic disease, there are other facts which point towards the probability of syphilis, notably the condition of the pupils, and in one of the cases the large number of deaths in infancy.

(5) In a case which will be referred to later (p. 568), the only explanation which could be found to account for the absence of the jerks was previous syphilis, and in this case there was a definite history of the disease.

(6) It has been pointed out by Babinski[1] that the Achillis-jerk is often lost before the knee-jerk in tabes.

One Hundred Cases of Nervous Diseases.

The Achillis-jerks were examined in 100 patients suffering from various forms of nervous disease under the care of Dr. Byrom Bramwell. The following table specifies the particular diseases in question, while a separate column shows the number of cases in which either one or both

[1] *Revue Neurologique*, 1901, p. 482.

Achillis-jerks were absent, or greatly diminished in activity:—

	No. of Cases.	Achillis-jerks lost or greatly diminished.
Disseminated sclerosis	13	0
Hysteria	11	0
Intracranial tumour	10	0
Peripheral neuritis	9	6
Chorea	9	0
Epilepsy	8	0
General paralysis of the insane	4	1
Hemiplegia	4	0
Sciatica	4	2
Spastic paraplegia	4	0
Neurasthenia	3	0
Spinal caries	3	0
Jacksonian epilepsy, auditory vertigo, local neuritis of upper limb, infantile paralysis,* syringomyelia, and Bell's palsy*—2 cases of each	12	2*
Pseudo-hypertrophic paralysis,† tubercular meningitis, miner's nystagmus, cerebral syphilis, lesion of cauda equina,† and a case of defective mental development —1 case of each	6	2†
Total	100	13

It will be seen from this table that the Achillis-jerks were present and marked in all but thirteen cases. Either one or both Achillis-jerks were absent or greatly diminished in six cases of peripheral neuritis, in two cases of sciatica and in one case of each of the following conditions, viz., pseudo-hypertrophic paralysis, infantile paralysis, a lesion of the cauda equina, general paralysis of the insane and Bell's

palsy. It will be convenient to refer firstly to these single exceptional cases, considering afterwards the state of the Achillis-jerks in sciatica and in peripheral neuritis.

Case 1.—A. H., schoolboy, aged 11, suffering from *pseudo-hypertrophic paralysis*. Both the knee-jerks and Achillis-jerks were present, although greatly diminished in activity and somewhat difficult to obtain. The state of the jerks in this case was only what was to be expected from the *weak condition of the leg muscles*.

Case 2.—J. G., van-driver, aged 24, suffering from the results of an attack of *acute anterior poliomyelitis*. The right Achillis-jerk was absent, the left present, the right knee-jerk being active, while the left was absent. *The wasted condition of the right calf* and left thigh muscles was obviously responsible for the absent jerks in this case.

Case 3.—D. H., labourer, aged 48, was admitted to hospital suffering from a *lesion of the cauda equina*. His illness had commenced six years previously with pains in the legs, and the condition had progressed for twelve or fifteen months, since which time his symptoms had remained *in statu quo*. A definite history of syphilitic infection followed by characteristic secondary symptoms was obtained. The history and distribution of his symptoms at the time of examination pointed to a lesion involving the sacral roots. Neither Achillis-jerk was obtained, the left knee-jerk was difficult to get, the right was absent. In this case, *the great wasting of the calf muscles* afforded an obvious explanation to account for the lost jerks.

Case 4.—J. R., an old soldier, aged 34, with a typical Bell's palsy of ten days' duration. On testing the Achillis-jerks, it was found that only the slightest movement of either foot was obtained even with reinforcement. The knee-jerks were present but feeble. The pupils reacted well to light and a thorough examination failed to reveal any signs suggestive of tabes, general paralysis or peripheral neuritis. There was no history of sciatica. The patient gave a definite history of hard sore some ten years previously, for which "black wash" had been ordered; there appeared to have been no secondary symptoms. *Syphilis* was probably the cause of the defective Achillis-jerk in this case.

Case 5.—G. K., aged 32, admitted to the R. I. E. suffering from *general paralysis of the insane*. His symptoms, which were sufficiently typical to permit of a positive diagnosis, need not

detain us. The knee-jerks were abnormally active, the left Achillis-jerk was absent and the right distinctly diminished. This patient had had *syphilis* followed by secondary symptoms.

The Achillis-Jerks in Peripheral Neuritis.

It will be seen from the table on page 567 that nine cases of peripheral neuritis were examined. Of these cases seven were cases of alcoholic neuritis.

In four cases of alcoholic neuritis (one man and three women), both the knee and Achillis-jerks were absent.

In the three remaining cases the knee and Achillis-jerks were present; in one case the knee-jerks were exaggerated. These three patients were all males, all had been drinking heavily, and in all there were pains in the legs and marked tenderness of the calf muscles. With regard to the classification of these cases, the objection might be raised that the evidence upon which the diagnosis of neuritis was based is not sufficiently strong. Even if it be granted that this is so, their inclusion seems justifiable in order to show that pains in the legs and marked hyperalgesia of the calves may exist as a result of alcoholism in association with normal Achillis-jerks.

In the case referred to on page 563 the condition would appear to have advanced a stage further, for in that case the Achillis-jerks were both absent.

Although from a single case such as that just mentioned one would hesitate to make the general statement that the Achillis-jerks are lost, or sometimes lost, before the knee-jerk in alcoholic neuritis, since there is always the possibility of some additional unrecognised etiological factor having been present, still the loss of the Achillis-jerks in this case *suggests* that such may be the case.

Two cases classed as peripheral neuritis still remain for consideration.

The first case is that of a sailor, aged 41, who was shown to the Medico-Chirurgical Society of Edinburgh by Dr. Byrom Bramwell.[1] The patient was suffering from a peculiar form of muscular atrophy of *peripheral distribution*

[1] *Trans. Med. Chirurg. Soc. of Edinburgh*, 1901, p. 146.

commencing in the lower limbs and afterwards affecting the upper. The onset was gradual and associated with cramps in the muscles. There was no objective disturbance of sensation and no affection of the bladder. The knee-jerks were feeble and the Achillis-jerks absent on both sides. *The absence of the Achillis-jerks was obviously due to the great wasting of the calf muscles* (all power of movement was completely lost in all the muscles below the knee on both sides). The diagnosis in this case was somewhat uncertain. Beri-beri was considered as a possibility, but finally excluded. In some respects the case resembled the peroneal type of muscular atrophy. A very similar case has been described by Mills.[1]

In a second case, which was classed as peripheral neuritis, both the Achillis-jerks were absent, while the knee-jerks were present though sluggish. This case presented many peculiar features, and the etiology of the condition was quite obscure. Space does not, however, permit of a detailed description of it.

The Achillis-Jerks in Sciatica.

The Achillis-jerks were examined in four cases of sciatica; in two of these cases they were affected and in two normal. The clinical features of the two cases in which the jerks were affected were briefly as follows:—

Case 1.—A labourer, aged 30, was admitted to hospital suffering from left-sided sciatica; the left Achillis-jerk was absent, the right although present was distinctly diminished; the knee-jerks were equal and of about normal activity. Upon enquiry, it was ascertained that, three years previously, he had had a severe attack of sciatica on the *right* side.

Case 2.—A farmer, aged 36, was admitted to hospital suffering from left-sided sciatica; the left Achillis-jerk was absent, while the right was difficult to obtain. Both knee-jerks were about normal, the left perhaps a shade less marked than the right. This patient had not previously suffered from sciatica.

It was pointed out by Babinski, in 1896, that in cases of sciatica the Achillis-jerk is usually abolished on the affected

[1] *Journal of Nervous and Mental Disease*, 1900, p. 324.

side. This observation has been confirmed by other observers. According to Max Biro[1] this alteration of the Achillis-jerk —abolition or diminution—which occurs in sciatica, is met with only on the affected, never on the sound side. The presence of the alteration of the reflex in a case of sciatica, indicates that the condition is in reality a neuritis of the sciatic nerve.

Max Biro[2] states that in cases of sciatica the lost reflex returns gradually with recovery. It appears, however, from one of the cases described above that years may elapse before the normal activity of the jerk is regained.

In the case of J. C., referred to on page 562, in which the loss of both Achillis-jerks could only be accounted for by two separate attacks of sciatica, the Achillis-jerks were both absent when the patient came under observation two years later.

The absence of the Achillis-jerk must be regarded as a valuable sign in distinguishing true sciatica from the hysterical variety.

The Achillis-Jerks in Tabes Dorsalis.

The writer has examined the Achillis-jerks in thirty cases of tabes seen in the hospital practice of his father.

In twenty-five of these cases both the knee-jerks and Achillis-jerks were absent.

The condition of the jerks in the five exceptional cases was as follows :—

(1) Both knee- and Achillis-jerks increased.

(2) Both knee- and Achillis-jerks present and about normal.

(3) Both knee-jerks and one Achillis-jerk active, the other Achillis-jerk constantly diminished.

(4) Both knee-jerks active, one Achillis-jerk absent, the other slight.

(5) Both knee-jerks and one Achillis-jerk absent, the other Achillis-jerk being active.

Thus of four tabetics in whom the knee-jerks showed no

[1] *Deutsche Zeitschrift f. Nervenheilkunde*, 1901, S. 188. [2] *Loc. cit.*

diminution, in two the absence or constant diminution of the Achillis-jerk afforded diagnostic data; in one of these cases the information derived being of undoubted value in substantiating the diagnosis. The following is a brief record of this case:—

J. C., aged 40, a sailor, complaining of double vision which had lasted for six months. The patient had had *syphilis* twelve years previously, followed by secondary symptoms. He had not had sciatica.

For three or four months he had had *occasional slight shooting pains in the legs.* There had been some *delay in micturition*, and he stated that occasionally the urine dribbled away at night. The water comes in a good stream. He had had some *diminution of sexual desire and power* during the last few months.

On examination, it was found that there was *complete paralysis of the right third nerve and that neither pupil reacted* to light. There was no ataxy and no Rombergism. No analgesia was detected in the arms, legs or trunk. The knee-jerks were brisk and equal. *The right Achillis-jerk was marked, the left distinctly sluggish.* There was a double flexor response. His memory was good and no mental peculiarity was noticed. The optic discs were well coloured and their edges sharp. There was no tremor of the tongue or hands, no twitching about the face and no affection of speech.

In the great majority of tabetics both the knee- and Achillis-jerks are lost at the time they first come to the physician.

Goldflam,[1] writing upon tabes in 1888, mentions incidentally the Achillis-jerk, which, he says, is generally abolished at the same time as the knee-jerk.

Ostankow[2] has recorded twenty-six cases in which he examined both the knee- and Achillis-jerks; in all of these cases both jerks were absent.

Babinski,[3] in the same year, stated to the Soc. Méd. des Hôp. de Paris, that in tabes the Achillis-jerk is sometimes lost before the knee-jerk, and that the absence of the Achillis-jerk will help the diagnosis in such a case. He had met with two cases in which the knee-jerks were lost while the

[1] Goldflam, *Neurolog. Centralblatt*, 1888, S. 528, u. 556.
[2] Ostankow, *Neurolog. Centralblatt*, 1898, S. 140.
[3] *La Semaine Médicale*, 1898, p. 439.

Achillis-jerks were still present; on the other hand, he had seen three cases where the Achillis-jerks were absent, and the knee-jerks present.

Mills,[1] in a paper read before the New York Neurological Society, January 3, 1899, refers to a case of tabes in which the absence of both Achillis-jerks "was of decided value in reaching a correct diagnosis." "This was a case sent to me for opinion by two physicians. The diagnosis of neurasthenia had been given, and the existence of tabes was regarded as in doubt, chiefly for the reason that both knee-jerks were present, although the patient had a recent history of what appeared to be shooting pains, and some evidences of probable slight involvement of the bulbar nuclei. Ataxia was not present, but the tendo Achillis-jerk was lost on both sides, quadriceps-jerk, knee-jerk, and gastrocnemius-jerk being present. I concluded that the case was organic, probably a somewhat irregular form of tabes."

Babinski[2] states that since his attention has been especially directed to the question of the Achillis-jerk in tabes, he has observed five cases of affection of the knee-jerk with a normal Achillis-jerk in that disease, whereas he has seen forty patients (*une quarantaine de malades*), undoubted tabetics, in whom the Achillis-jerk was alone affected, or was affected to a greater degree than the knee-jerk. He has come to the conclusion that in the first stages of the disease the Achillis-jerk affords data for diagnosis more frequently than the knee-jerk.

Although the literature upon the subject is as yet small, the above observations will serve to show that *the Achillis-jerk should always be examined in a suspected case of early tabes in which the knee-jerks are still present, and that in such cases its absence may be of considerable value in making the diagnosis.*

The loss of the Achillis-jerk in general paralysis is, no doubt, to be accounted for by an associated "tabetic" lesion; it may prove of value in the diagnosis of early cases of this disease.

[1] *Journal of Nervous and Mental Disease*, 1899, p. 159.
[2] *Revue Neurologique*, 1901, p. 483.

Table Illustrating the State of the Achillis-jerk in 1,009 *Cases of Health and Disease.*

	Class of Case.	No.	One or both Achillis-jerks absent or greatly diminished.	Remarks regarding cases where Achillis-jerks were absent or diminished.	Cause of loss or diminution of Achillis-jerks.
1	Sick children	50	0		
2	School ,,	219	4	All boys and all deaf-mutes	?
3	Fever convalescents	100	3	2 Post-diphtheritic paralysis	Post-diphtheritic neuritis
				1 Convalescence from scarlet fever	Leg muscles not properly relaxed
4	Medical students	80	0		
5	Insane ..	100	11	5 General paralysis ..	G.P.I.
				5 Senile dementia ..	Senility
				1 Alcoholic insanity ..	Previous alcoholic neuritis
6	Persons over 40 years of age from Workhouse Population	230	86	All over 50 years of age. Frequency of absence increases with increasing years (see Table, p. 559)	Senility
7	Hospital Cases (General Medical)	100	8	2 Diabetes mellitus ..	Diabetes mellitus
				1 Dyspepsia	Early alcoholic neuritis
				1 Dyspepsia	Influenza?
				1 Gout	Previous sciatica
				2 Aortic aneurism 1 Aortic incompetence	Syphilis?
8	Hospital Cases (Nervous, exclusive of tabes)	100	13	6 Peripheral neuritis	Peripheral neuritis
				2 Sciatica	Sciatic neuritis
				1 Pol. ant. acuta 1 Pseudo-hypertrophic paralysis 1 Lesion of cauda equina	Atrophy of calf muscles
				1 Bell's palsy	Syphilis
				1 G.P.I.	
9	Hospital Cases (Tabes dorsalis)	30	28	25 Both knee-jerks and Achillis-jerks absent	Tabetic degeneration of afferent neurons
				1 Knee-jerks active; right Achillis-jerk absent; left Achillis-jerk slight	
				1 Knee-jerks active; right Achillis-jerk active; left Achillis-jerk constantly diminished	
				1 Knee-jerks absent; right Achillis-jerk active; left Achillis-jerk absent	
	Total	1009	cases		

Conclusions.

(1) The Achillis-jerk is most easily and conveniently obtained in the kneeling position. The examination in the prone position is more difficult and requires considerable experience, although the results obtained in this position are almost equally reliable in the hands of a skilled observer.

(2) The Achillis-jerk is (probably) constantly present in "health" in persons under 50 years of age. In other words the loss of the Achillis-jerk in a person under 50 years of age (in the absence of any local cause, such as great œdema of the legs, which may mechanically interfere with the examination) is always to be looked upon as *a sign of organic disease*, with the same assurance as loss of the knee-jerk is so regarded at the present time.

(3) In persons over 50 years of age the activity of the Achillis-jerk diminishes with increasing age (see table, p. 559), consequently in old people absence of the Achillis-jerk, especially if bilateral, cannot be regarded as having the same diagnostic significance that it has in children, young persons and middle-aged adults.

(4) In the majority of cases of disease in which the Achillis-jerks are absent, the knee-jerks are also lost, *e.g.*, most cases of tabes, peripheral neuritis, &c.

(5) In certain conditions in which the knee-jerks are unaltered the Achillis-jerks are found to be absent or greatly diminished. Under these circumstances loss of the Achillis-jerk may prove a valuable adjunct to diagnosis.

(6) Since in tabes the Achillis-jerk is usually lost before the knee-jerk, its condition should always be ascertained in suspected cases of that disease (Babinski), and more particularly in those cases in which the knee-jerks are present.

(7) It has been shown by Babinski and others that in cases of sciatica the Achillis-jerk is usually lost on the affected side; the loss of the Achillis-jerk proves the existence of a sciatic neuritis. The absence of the Achillis-jerk is for this reason a physical sign of importance in the diagnosis between true sciatica and the hysterical form of the disease.

(8) The loss of the Achillis-jerk caused by a sciatic neuritis may persist for years after the sciatica has itself disappeared.

(9) Cases of multiple neuritis are met with in which the Achillis-jerks are absent, while the activity of the knee-jerks remains unimpaired. In such cases the absence of the Achillis-jerks is of value in diagnosis.

(10) It appears probable that the Achillis-jerk is sometimes lost as a result of syphilis, just as the Argyll Robertson pupil sometimes occurs as a result of that disease unaccompanied by other signs of tabes or general paralysis.

(11) The absence of the Achillis-jerk is sometimes of value in localising lesions in the spinal cord or the nerve roots.

ON THE SEAT OF THE PSYCHO-PHYSICAL PROCESSES.

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THE term "psycho-physical process" has been used with various meanings by different authors; it is important therefore to define at once the sense in which it is used in the title of this paper and throughout the following pages. By psycho-physical process I mean to denote a physiological process that is immediately correlated with and invariably accompanied by a psychical process. In this usage I follow Prof. G. E. Müller,[1] the distinguished psychologist of Göttingen, on whom, as regards all things psycho-physical, the mantle of G. T. Fechner may be said to have fallen. I would prefer to define it more fully as that part of the total process of physiological excitation within the nervous system which stands in a relation of immediate interaction with psychical process or consciousness. But this latter definition, involving as it does the rejection of the hypothesis of psycho-physical parallelism, would be unacceptable to the very numerous upholders of that doctrine, and the former is sufficient for the purpose of this essay. Now, whether we hold by the doctrine of psycho-physical parallelism or by the hypothesis of interaction between physical and psychical processes, we have to recognise that not all of the processes that constitute the activity of the brain at any moment of consciousness are psycho-physical processes in the sense defined above; it is,

[1] *Cf.* "Zur Psychophysik der Gesichtsempfindungen," § 1. *Zeitschrift für Psychologie*, Bd. X.

for example, generally admitted that the medullated fibres of the white matter of the brain and cord are entirely similar in nature and function to the fibres of the peripheral nerves, that their function is the transmission, and possibly the augmentation, of the state of excitation, and that this process is not immediately correlated with psychical process.[1] It becomes then a matter of the greatest interest to inquire in what part of the nervous system we must seek the psycho-physical processes.

The conclusion that the nerve-fibres of the white matter are not the seats of the psycho-physical processes has been extended by analogy and with a high degree of probability to all nerve-fibres, *i.e.*, to all processes of nerve-cells, whether they occur in the white or grey matter of the brain or cord, and, since it was generally held until quite recently, that the nerve-cells and their processes constitute the whole of the essentially active parts of the nervous system, the conclusion that the bodies of the nerve-cells or of some of them are the seats of the psycho-physical processes seemed to be inevitable, and has in fact been very generally accepted, though its acceptance has been perhaps more often implied than expressed. It must be admitted that this view has remained singularly barren up to the present time.

With the advent of the "neurone-theory," however, a new possibility was opened up, for it is of the essence of this theory that there is no continuity of substance between the individual neurones, and it seems therefore necessary to assume that the process by which one neurone communicates its excitement to another may be, and probably is, different from that which constitutes the passage of the nervous impulse along axones or dendrites. If, then, we accept the neurone-theory, the problem of the seats of the psycho-physical processes presents itself in the form of a dilemma; they may be seated either in the bodies of nerve-

[1] Even if we incline to believe that the movements of all matter, or of all living matter, are associated with some kind of consciousness, we shall still have to admit that the fibres of the peripheral nerves and of the white matter of brain and cord are not immediately concerned with the higher kinds of consciousness.

cells or at the places of junction of neurones, at synapses.[1] In a paper published some years ago [2] I ventured to present certain arguments in favour of the second possibility, to urge that we must regard the synapses as the seats of the psycho-physical processes. At that time there seemed to exist no histological basis for this view, for it was commonly taught that, besides the nerve-cells and their processes, there exists in the grey matter nothing but fluid plasma and other substances not immediately concerned with neural processes, the blood-vessels and the neuroglia a purely supporting tissue. It seemed to me, therefore, and perhaps to others also, a view somewhat wildly revolutionary and hard to be believed. But since that time further reading has shown me that the idea is hovering, as it were, in the neurological atmosphere, and that new evidence pointing in the same direction continually appears. Further, a possible histological basis has been described by a high authority, while certain observations made during a study of visual sensations and in part already published [3] seem strongly to support the view.

As my previous paper was written primarily for psychologists the argument was based chiefly on psychological grounds and the physiological evidence was not detailed. It seems worth while therefore to attempt to hasten the precipitation of this view and to give it currency by putting together in this paper as briefly as possible all that can be said in its favour as against the view that the bodies of the nerve-cells are the seats of the psycho-physical processes.

First let me point out that neither view is necessarily materialistic, that neither excludes the conception of an immaterial active soul. If the soul is a valid conception, then we must admit of the soul that it is where it acts or is acted upon, so far as our ideas of space are in any sense applicable to it. And if the view that I advocate should

[1] The name proposed by Foster and Sherrington for the junctions of neurones without continuity of substance. (See "Sir Michael Foster's Text-book of Physiology," vol. 3, p. 929. Seventh edition).

[2] *Mind*, N. S., vol. vii. "A contribution towards an improvement in psychological method."

[3] *Mind*, vol. X. "Some new observations in support of Thomas Young's theory of light- and colour-vision."

prove to be true, then those who adopt the spiritualistic hypothesis will have to regard the synapses as the places of interaction of soul and body; as Lotze writes in the course of an admirable discussion of this subject: "Wherever there are elements with which the nature of the soul enables and compels it to interact, there it will be present and active; wherever there is no such summons to action, there it will not be or will appear not to be."[1] And, in order clearly to define the philosophical standpoint from which my enquiry sets out, I will quote another passage from the same work of this distinguished author: "We shall never succeed in analytically deducing the feeling from the nature of its physical excitant; we can only connect the two synthetically; and the physical event does not become a condition of the rise of the feeling until the sum of motions in which it consists meets with a subject which in its own nature has the peculiar capacity of producing feeling from itself. In this fact a limit is at once placed to all physiological and psychological enquiry. It is utterly fruitless to attempt to show how a physical nervous process gradually transforms itself (as we are told) into sensation or any other mental occurrence. There remains only the different but extremely important task of discovering *what* psychical event a and *what* physical stimulus a are as a matter of fact universally connected in the order of nature, and of finding the law by which a undergoes a definite change and becomes β, when a by a change equally definite (but definable only by a physical standard and not a psychical one) becomes b."[2]

The evidence seems to fall naturally under four heads, namely, the anatomical, the purely physiological, that derived from the study of sensations (the ground on which it is impossible to separate physiology and psychology), and lastly the psychological.

The Anatomical Evidence.

As regards the structure of the bodies of nerve-cells, most histologists would seem to have returned now to the

[1] "Metaphysik," § 295. Bosanquet's translation. [2] *Ibid.*, § 238.

view of Max Schultze that the fibrils, which seem to be the conducting elements of the axones, run into and through (in some cases at least) the cell-bodies and out into the dendrites.[1] And many of those who reject this view yet regard the "non-stainable substance" of the cell-body as closely allied to, if not identical in structure with, the substance of the axone.[2] The "stainable substance" of the perikaryon,[3] the tigroid bodies of v. Lenhoessek and other writers, is regarded by high authorities as nutritive in function. Nissl,[4] in particular, to whom our knowledge of this substance is chiefly due, has shown that when, through the action of poisons, it undergoes very marked changes, these changes are not directly correlated with the disturbances of nervous and psychical functions. It seems doubtful whether the non-stainable substance of the perikaryon contains any other substance than that which so closely resembles the conducting substance of the axones, but if any such third substance is present it must be in very small quantity only. Again, that the nutrition of the axones is in some way dependent upon the cell-body is fully established. This dependence is in all probability a special case of the general law established by the researches of Verworn and others on unicellular organisms, the law that an interchange of substance between the nucleus and the extranuclear protoplasm is essential to the continued activity and life of both.[5] Now in the case of most neurones the mass of the substance of the cell processes, axones and dendrites, is very great in proportion to the mass of the cell-body. Donaldson calculates that in some cases the volume of the axis-cylinder-process alone is as much as 187 times the volume of the cell-body. These considerations alone should make us hesitate to regard the cell-bodies as the seats of any other processes

[1] *Cf.* Schaefer. Art. "Nerve cell," "Text-book of Physiology," p. 596. Edinburgh, 1900.

[2] *Cf.* L. F. Barker. "The nervous system and its constituent neurones," p. 136. London, 1900.

[3] The name suggested by Foster and Sherrington for the substance of the cell-body surrounding the nucleus.

[4] Nissl. "Nervzellen und graue Substanz." Part 1. *Münchener medizinische Wochenschrift*, Bd. 45, 1898.

[5] *Cf.* Max Verworn. "Allgemeine Physiologie." Jena, 3rd Ed., 1901.

than those concerned in the conduction of the impulses and in the nutrition of the whole cell, and least of all can we assign to them the psycho-physical processes. For we must assume that these processes are the most complex and delicate of all that occur in the animal body, and for each primary sensation-quality at least we must assume a specifically differentiated psycho-physical process, involving in all probability complex chemical changes, and therefore a complex material substratum.[1] It may then be said with some confidence that there is no histological evidence of a sufficient material substratum for the psycho-physical processes within the cell bodies.

How then does the matter stand with the synapses? As to the relation of the terminals of fibrils with those of other fibrils or with the cell-bodies at synapses, it was, until very recently, the generally accepted view that there is a simple apposition and no continuity of substance, and many interesting speculations have been advanced regarding the possible changes of relation of these terminals conditioning changes of functions.[2] This view of the lack of continuity was supposed to be true of all junctions of neurones, and its universal application seemed to be generally held as an essential part of the neurone-theory. But during the last few years doubt has been thrown upon this view. Continuity of substance has been proved to exist in certain instances, and with that tendency to generalise from a few particulars so curiously prevalent among histologists, some have concluded that the establishment of these instances of continuity deals the neurone-theory its death-blow. Of observations of this kind only the more important need be noticed. Dr. Alex. Hill has described a continuity of neurones by means of fine core-like threads stretching across from fibril to fibril and more or less sheathed in a stainable plasma, and he suggests that the relation of this plasma-sheath to the core is variable and conditions the

[1] For an interesting discussion of the probable nature of the psycho-physical processes see G. E. Müller, *loc cit.*, § 9.

[2] For a summary of these speculations and of the evidence for amœboid movements of terminals, see Duval "L'amoeboidisme du systeme nerveux." *Revue Scientifique.* Paris, 1898.

degree of conductivity or intimacy of functional union between the neurones.[1]

It is clear, of course, that if this kind of union be shown to be of general occurrence, the neurone-theory will not be weakened but rather strengthened, inasmuch as our conception of the relations of neurones to one another and of the constitution of the synapses will be clearer and more definite.

Held has shown that in the case of many of the cells in several of the nuclei of the pons, in the cortex of the cerebellum and in the anterior horns of the cord of the rabbit, it is possible to trace a growing intimacy of union between terminals of axones and cell-bodies. About the time of birth of the animal there is simple proximity of a group of fibrils, the terminals come into contact with and gradually fuse with the cell-body by a process that Held calls "concrescence." For some time the junction remains marked by a layer of more highly refractive substance, but in some cases this too disappears and the fibrils which may penetrate far into the cell-body can only be distinguished from it by their slightly different texture and staining-reactions.[2] Apathy has described in the ganglia of the leech and earthworm a state of affairs still more remote from the discontinuity usually postulated as the basis of the neurone-theory, namely, a fibrillar network passing without breach of continuity between and through the bodies of several nerve-cells.[3]

These observations by Held and Apathy seem to be not only not incompatible with the neurone-theory but rather to prove the existence of just such a state of affairs as is demanded by that theory when conceived in its only rational form. In the paper referred to above[4] I ventured to sum up the phylogeny of the nervous system as a process of constant proliferation of new neurones at the top of the

[1] A. Hill. "Note on thorns and a theory of the constitution of the grey matter." BRAIN, vol. xx., 1897.
[2] H. Held. "Beiträge zur Structur d. Nervenzellen." *Achr. für Anat. u. Phys.* 1897. *Anat. Abth.*, Heft 3 and 4.
[3] Mittheilung aus. d. Zool., Station zu Neapel, Bd. 12, 1897.
[4] *Mind.* New series, vol. vii.

system with a progressive integration of the older neurones of the lower (*i.e.*, older) levels into conduction-paths of increasing fixity, *i.e.*, of increasing intimacy of union of the constituent neurones, the intimacy of union being a function of the degree of organisation of the synapses. If there be any truth in this scheme we must expect to find, in the nervous system of any progressive species of vertebrate animal, all degrees of intimacy of union of neurones, from actual continuity in the cord and lower parts of the brain to mere proximity of terminals in the case of many synapses of the cerebral cortex, and we should expect to find a marked difference in this respect between the very young and the old animal.

We should expect also to find a maximum degree of fixity of conduction-paths, of intimacy of union of neurones, in the nervous systems of some of the lowly and unprogressive types of invertebrate; for, as I pointed out, in the case of a lowly species that has followed for thousands of generations a simple mode of life in a simple environment, "it may be reasonably supposed that its nervous system is completely organised to carry out automatically its few motor reactions to the comparatively simple impressions made upon its sensory nerves." Held's observations show us how, as a matter of fact, this phylogenetic process of organisation of neurones into fixed conduction-paths is recapitulated in the case of some at least of the neurones of the lower parts of the brain of the rabbit, while in the continuous networks running through cell-bodies in the ganglia of the leech, as described by Apathy, we may see conduction-paths of the highest degree of organisation, actual continuity of conducting substance implying a corresponding fixity of function. Unfortunately Held does not seem to have looked for concrescence between neurones of the cortex. It must be noted that Held does not regard his discoveries as in any way invalidating the neurone-theory, and that, at the end of his paper, he points out that the substance of the concrescence-surface is probably of high importance, that it is probably the part of any conduction-path most readily affected by poisons, and may play a part

in the determination of sleep, fatigue and attention and of the variable lost time of reflexes.

The theory of the individuality and discontinuity of neurones must not, then, be held too rigidly. We must rather believe that while all are originally discontinuous they tend phylogenetically to become more and more intimately united into series to form conduction-paths of greater and greater fixity, a process that is recapitulated and carried further in the individual as the result of use.

And the neurone-theory must be modified in another important particular; we may no longer assume that the neurones, the cell-bodies and their processes, constitute the whole of the essentially nervous or active substance and that the only substances that lie between and around them are the blood-vessels, neuroglia, and lymph, merely supporting and nutritive in function.

For Nissl has recently brought forward positive evidence of a substance different from these, existing outside and between the neurones, a substance which is especially abundant in the cerebral cortex of man.[1] He writes: "the significance of the newer discoveries consists in this, that the nervous tissue may no longer be conceived as hitherto, as a community of numberless cell-individuals, but we must think of the nervous tissue as consisting of nerve-cells *and* a specific nervous substance, namely, the fibrillar substance. This, if I am not mistaken, is to be conceived as a living substance that is derived from the protoplasm of the nerve-cells and doubtless stands to the nucleus-holding cell in a relation of intimate dependence, but which, in spite of that, must be also in other respects in a very considerable degree independent of the cells, as is shown by the fact that in great part it develops and maintains itself external to the cell. This fibrillar substance seems, according to the latest discoveries, to be the carrier of the nervous functions. It represents therefore the highest step of differentiation of the cell-plasma within the animal body." Nissl points out that this inter-cellular substance, and not, as is commonly assumed, the presence of many cell-bodies and dendrites, is

[1] F. Nissl. *Loc. cit.*, three papers.

the specific characteristic of the grey matter which distinguishes it from the white matter.

The principal evidence for the existence of this inter-cellular substance is that "the higher the grade of development of an animal is, the fewer nerve-cells inhabit an equally large volume of cortex"; and that in the human cortex it is impossible to account for the whole mass of the grey matter as nerve-cells and their processes and neuroglia, for, when the mass of these is subtracted, there remains, especially in the middle layers of the cortex, a very considerable mass of substance not accounted for. As to the structure or arrangement of this substance Nissl's statements are not very definite, but he inclines to regard it as fibrillar. Here then we have histological evidence of an abundant inter-cellular substance that may be regarded as the seat of the psycho-physical processes, or if anyone prefer the expression, as the material basis of consciousness, while in the case of the cell-bodies, as we have seen, there is no such evidence.

The Physiological Evidence.

We know that there are various states of the central nervous system characterised by differences in the degree of resistance offered to the propagation of the nervous impulses from the afferent to the efferent nerve-fibres, of which states sleep and waking are the most familiar. That the synapses are the seats of these changes in the degree of resistance has appeared probable to many writers.[1] We know also that, in passing through the central nervous system, the impulses are delayed, there is occasioned in some way what is known as the "lost time." Thirdly, we know that some part of the central conduction-path acts like a valve, permitting the passage of impulses from the afferent towards the efferent side, but preventing entirely, or at least offering great resistance to, passage in the reverse direction. Fourthly, we know that a very rapidly repeated or a continuous stimulus thrown into an afferent nerve or into the motor cortex

[1] *Cf.* Duval. *Loc. cit.*

issues in the efferent nerves as a regular series of impulses at the rate of about ten to nineteen per second, and that in voluntary effort also a similar series of impulses issues along the efferent nerves.

It is perhaps worth while briefly to resume the evidence that all these effects, the varying resistance, the lost time, the valve-like blocking of impulses, and the transformation of the continuous excitation, or rapidly repeated series of excitations, into a series of moderate rate, are determined at the synapses and not at the cell-bodies; for here again there seem to be only two possibilities—the problem appears as a dilemma—cell-body or synapsis.

In evidence against the cell-body we have the following facts: firstly, it seems to be established, as was noted above, that in many cases, if not in all, the fibrils of the cell-processes run through the cell-bodies without interruption, and there seems to be no direct evidence of any sort to show that the bodies of cells offer any resistance to the passage of impulses through them; we know on the other hand that in passing through a spinal ganglion the impulse suffers no appreciable time delay,[1] and that impulses, or at least electric changes such as always accompany a nerve-impulse, may be transmitted through the spinal ganglia in both directions;[2] lastly, we know from Bethe's remarkable and oft-quoted experiment on *Carcinus Mænas* that a nerve-fibre deprived of its cell-body may continue for some time to play its part in producing reflex movements in an apparently normal manner.

That the synapses are the seats of the valve-effect and are therefore at the root of what has been called the "law of forward-conduction of nerve-impulses," and that they occasion the lost time of reflexes and simple reactions, are propositions now pretty generally accepted. The former seems to be proved by the experiments of Gotch and Horsley, which showed that the nerve-impulse may be transmitted in the reverse of the normal direction through the spinal ganglia and through the efferent nerves, but is not, in the latter

[1] Schäfer. Art. "Nerve Cell," "Text-book of Physiol.," p. 604.
[2] Gotch and Horsley. *Phil. Trans.*, vol. clxxxii. B.

case, passed on to other neurones of the cord; while the latter is rendered highly probable by the simple process of excluding the alternative, the cell-body, no lost time being demonstrable in the case of the spinal ganglia. There is less agreement as to the seat of the determination of the rhythmic series of efferent impulses and the variations of resistance.

Schäfer, to whom we owe so much of our knowledge of the discontinuous rhythmic character of the efferent outflow, writes: "This rhythm must in all probability be set up in the motor nerve-cell," and he seeks to bring it under one point of view with the determination of the "lost time" by suggesting that every neurone has a latent period of excitation analogous to that of striped muscle-fibres.[1] But this hypothesis cannot, I think, be made to fit with the facts. The first difficulty that suggests itself is this—If the rhythmic discharge of efferent impulses at ten per second is to be explained by assuming a latent period of excitation for the neurones, we shall have to assume that the duration of this latent period is approximately 100 σ[2] for at least one neurone of the conduction-path concerned in the rhythmic discharge. For we cannot suppose that small latent periods of the individuals of a chain of neurones will be summed together to give the appearance of a long latent period in the last of the chain, except in the case of a single stimulus or the first of a series of stimuli; the period between any two impulses of a rhythmic series determined in this way could not be longer than the period of latency of that neurone of the chain which has the longest latency. Now a reflex contraction, or one produced by stimulating the cross-section of the cord, exhibits a rhythm similar to that of contractions determined from the cortex,[3] yet, as Windt has shown, the total lost time of a cord-reflex may be as small as 8 σ, and, as Schäfer points out,[4] the lost time at each

[1] *Cf.* Schäfer on "The Block Between Nerve-cells." *Op. cit.*, p. 613.

[2] σ is the sign commonly used by psychologists to denote $\frac{1}{1000}$ sec., and will, I think, conduce to clearness if all brief periods are given in terms of σ

[3] Sherrington, "The Spinal Cord." "Schäfer's Text-book of Physiol. p. 831.

[4] *Op. cit.*, p. 609.

synapsis of a reflex conduction-path in the cord is probably only 2 σ. The latent period of the neurones of the cord, therefore, cannot be more than about 8 σ and probably not more than 4 σ, and therefore cannot account for a rhythmic interval of 100 σ.

The assumption of a latent period of excitation proves equally unsatisfactory when applied to the explanation of the lost time of reflex actions and of voluntary reactions. If such a latent period obtains, all analogy would lead us to expect that it should be approximately of the same duration for different neurones and for the same neurones at different times. But this is very far from being the case. In the first place, the lost time of a simple reflex is shorter the stronger the stimulus, and with a sufficiently strong stimulus may become inappreciable;[1] and in the case of simple reactions, too, the lost time is in some cases shorter with stronger stimulus.[2] Again, the delay at the synapses must be greater for most of the synapses of the cortex than for those that occur in the course of reflex conduction-paths of the cord. The lost time at a synapsis of the latter kind cannot be more than about 4 σ, perhaps not more than 2 σ,[3] yet the lost time of the cortex on direct stimulation of its surface is found to be about 20 σ, and this represents in all probability the lost time of a single synapsis, or at most of two. Again, the lost time of a single "motor" reaction may be exceeded by that of a simple "sensorial" reaction by 100 σ, and by that of a "choice" reaction by as much as 400 σ.[4] If then we are to suppose the lost time at all synapses to be approximately equal we shall have to assume that in such a "choice" reaction the excitation runs through a chain of 100 to 200 neurones of the cortex successively, a conclusion that cannot be accepted for a moment. We must believe rather that in the reflex conduction-paths of the cord, paths of a high degree of constancy of function, the synapses are very thoroughly organised, *i.e.*, that their degree of re-

[1] Rosenthal, quoted by Exner. *Entwurf z. physiol. Erklärung d. Psych. Erscheinungen*, pp. 48 and 55.
[2] Exner. Hermann's "*Handbuch d. Physiologie*," Bd. ii.
[3] Schäfer. *Loc. cit.*, p. 609.
[4] Külpe. "Outlines of Psychology," p. 419.

sistance has been reduced to a minimum by frequent repetition of the particular reflex action in the individual and in the race, while in the higher parts of the nervous system the resistance, and therefore the loss of time, occasioned by the synapses is greater in the inverse order of their degree of organisation. This conclusion is borne out by the study of the effects of practice in shortening reaction-times. It is well known that the simple reaction-time may be somewhat shortened by practice until it becomes a purely automatic or reflex act, and in a similar manner reactions involving more complex mental processes, such as discrimination and choice, may on frequent repetition become more and more automatic in character, *i.e.*, they are performed more easily and regularly and with less and less clear conscious accompaniment, until they, too, resemble rather a reflex action, psychical activity being reduced to a minimum. And parallel with this progressive loss of psychic accompaniment goes a progressive shortening of the time lost at the synapses.

This shortening of the lost time at the synapses by repetition of the passage of the excitation across them may be best conceived as due to a diminution of the resistance offered by that delicate and complex inter-cellular substance which, as it seems to me, we have to regard as the seat of the psycho-physical processes.

The consideration of how exactly the various and variable periods of lost time are determined I postpone to the concluding section of this paper, but will adduce here two lines of evidence bearing out the view that the principal factor is the degree of resistance offered by a specific inter-cellular substance. Such evidence is afforded by the consideration of the action of certain drugs. The observations of Langley [1] and of Langley and Anderson [2] on the effects produced by nicotine on nerves and ganglia seem to show that nicotine, in paralysing a sympathetic ganglion, acts upon the junctions of the fibrils with the cells of the ganglion, and this in far smaller quantities than will appreciably affect nerve-cell or fibre. This implies either a differentiation of the terminals of the fibrils or the exist-

[1] *Journ. of Phys.*, vol. xi. [2] *Ibid.*, vol. xiii.

ence of a specific inter-cellular substance peculiarly subject to the paralysing action of nicotine.[1] The junctions of nerve-fibres with the cells of glands and muscles are also peculiarly easily affected by drugs and by fatigue, and it is not improbable that the constitution of such junctions is similar to that of the junctions of the neurones.

It is well known that ether, chloroform and alcohol, when applied directly to exposed nerves, will abolish their conductivity. But, if taken into the blood, they abolish the higher functions of the brain when present in such small quantities only as to leave the lower nervous mechanisms unaffected. I have elaborated this point in the paper before referred [2] to, and will therefore only briefly resume the argument as follows :—The functioning of the conduction-paths of the central nervous system seems to be abolished by an increasing quantity of any one of these drugs, strictly in the inverse order of the fixity of function of the paths, *i.e.*, of the degree of organisation of the synapses of the paths. Thus the extremely variable and complex and delicately-balanced processes involving self-consciousness are first diminished and then abolished ; and these are followed in turn by the more stable, more habitual functions, *i.e.*, those subserved by relatively well-organised conduction-paths ; while only in the deepest stages of the anaesthesia do we see affected the deep-seated vital reflexes, those whose conduction-paths, being constantly at work throughout life, have synapses of the highest degree of organisation. This is just the series of effects that must result if these drugs act first and chiefly upon the inter-cellular substance at the synapses, slowing or paralysing its metabolism in some degree and so raising its normal resistance. For where the resistance is normally greatest there complete blocking of the conduction-path must earliest result, and where it is least there conductivity will persist longest, the synapses will remain longest permeable to the nervous impulse.

The waste products of muscular activity exert an effect

[1] Recent observations by Langley (*Journ. of Phys.*, vol. xxvi.) make this conclusion seem less certain, but by no means disprove it.

[2] *Mind.* N. S., vol. vii.

on the nervous system comparable in many ways to that of anæsthetic drugs such as alcohol and ether, and it seems probable that they too may act principally on the intercellular substance increasing the resistance of the synapses. Especially noteworthy is it that fatigue, like alcohol, prolongs the lost time of reactions.[1] But however this may be, there is evidence that the synaptic processes are peculiarly liable to become fatigued when any conduction-path is continuously stimulated. The evidence is of two kinds, the one kind, afforded by certain observations on the behaviour of visual sensations, belongs to the following section, the other may be dealt with at once, with the proviso that its full force cannot be made clear till the former also has been presented. In a recent paper Mr. A. Levy[2] describes the fatigue effects that appear on repeatedly stimulating one spot on the motor cortex, the curve of contraction of the corresponding muscles being registered on a revolving drum. He finds that, when a faradic current is continuously applied, the contraction-curve of the muscle is maintained at a constant height for a period varying from 8 to 30 seconds and then falls suddenly, sometimes with a few clonic contractions, to the base line, and that then for a short period no contraction can be evoked even by a rather stronger stimulus applied to the same spot on the cortex. Levy writes: "It is difficult to regard this as evidence of anything else than that the motor cells have become fatigued."

Now we know that neither nerve-cells nor fibres in the peripheral system can be made to show any symptoms of fatigue at all comparable to this; and we know also that there are many reflex conduction-paths by means of which muscles may be kept in a state of contraction for very considerable periods with little or no signs of fatigue. A good instance of such long-continued activity of a reflex conduction-path is supplied by the carrying of a heavy pipe tightly clenched between the teeth while one remains unconscious of, or at least quite inattentive to, its presence. Now, between such a reflex conduction-path as is at work in

[1] Exner. *Hermann's Handbuch*, Bd. 2.
[2] *Journ. of Phys.*, vol. xxvi., "Fatigue of cerebral cortex," 1901.

this instance and one involving neurones of the cortex, the only differences, so far as we know, are firstly, that the former has by constant use become thoroughly organised so that the resistance of the synapses has been reduced to a minimum, while in the latter there must usually occur between cortical neurones synapses but little organised and of high resistance; secondly, that consciousness usually accompanies the activity of the paths of the latter kind, and not all, or only in some very obscure fashion, and in a minimal degree, the activity of paths of the former kind. It seems probable therefore that the sudden drop of the contraction-curve in Levy's experiments is due to fatigue, not of the motor-cells, but of the synaptic processes which effect the transmission of excitation from neurone to neurone, the resistance of the inter-cellular substance being so increased by this fatigue that the impulses set up in the superficial neurones of the cortex cease to be transmitted to the efferent neurones. Levy reports in the same paper other observations that support this view. He shows that with continued faradic stimulation, the sudden drop of the contraction-curve occurs earlier with a weak stimulus than with one of moderate strength, so that he is led to write: "It may then be concluded that cortical fatigue is more rapid in its progress as a result of weak stimulation." This, if true, would surely be a very extraordinary state of affairs; it is well-nigh incredible that any function should be fatigued more rapidly by a low than by a higher degree of activity. But if, as I suggest, the fatigue is synaptic and results in an increase of the normal resistance of the synapses, then it is obvious that the passage of a weak excitation may be barred by a smaller degree of fatigue, a smaller increase of the normal resistance than would be necessary to bar the passage of a stronger excitation.

Again, Levy shows that, if a faradic stimulation of about three seconds' duration be repeated at intervals of one second, the tops of the series of contraction-curves form a curve falling slowly and regularly and reaching the base line only after a very prolonged period of repeated stimulations. It appears, therefore, that the periods of rest of one second

suffice to prevent the onset of that peculiar form of fatigue which, under continuous stimulation, causes the complete and sudden fall of the curve after from eight to thirty seconds; and that, when this form of fatigue is prevented, there manifests itself an effect just such as would result from a fatigue of the neurones, *i.e.*, from a diminishing energy of response to the stimulations of constant strength. It seems then probable that in these experiments we have to do with two kinds of fatigue, namely, fatigue of the neurones that manifests itself in a gradual diminution of the energy of response, and fatigue of the inter-cellular substance characterised by its rapid onset and still more rapid disappearance.[1]

The Evidence from the Study of Sensations.

Certain phenomena that I have observed and in part described in a recent paper[2] seem to lend very strong support to the interpretation, suggested in the preceding section, of the fatigue-effects manifested by the motor cortex when directly stimulated. I find that if any dull or moderately bright patch of white or coloured light on a dark ground is fixated very steadily it is liable to undergo what I have called complete fading, *i.e.*, it will suddenly disappear from consciousness to reappear as suddenly and with undiminished brightness after an interval that varies from about half a second to several seconds. If the patch of light be upon a background that is not quite dark, but feebly illuminated, it happens not infrequently that the whole field of vision disappears, both the bright area and the background, leaving a sense of complete and extreme darkness, a darkness much

[1] V. Horsley (Brain, 1898) shows that if a continued faradic stimulus be thrown into the sciatic nerve, and the contraction curve of the reflexly excited crusens muscle of the same side be registered, the curve soon falls towards the base line. If then the other sciatic be stimulated the muscle contracts rapidly and strongly again. Horsley concludes, therefore, that the fall of the contraction curve is the expression, not of fatigue of the reflex-centre, but of some effect allied to inhibition. I suggest that here again we have to do with synaptic fatigue, namely, at the synapsis by which the afferent fibres of the sciatic nerve make contact with the reflex-centre.

[2] "Some New Observations in Support of Thomas Young's Theory of Light- and Colour-vision," Section i. *Mind*, vol. x.

more complete than is experienced on excluding all light from the eyes. This complete fading may occur during very steady fixation while both eyes are accommodated upon a near object, but it occurs far more readily if the accommodation be entirely relaxed and two similar bright images be combined by regarding them with the visual axes parallel. And it occurs most readily when the patch of light is fixated by one eye only (the other being closed and covered) with relaxed accommodation and a + 4 D glass before the eye to preserve definition and perfectly steady fixation.[1] A noteworthy fact is that sometimes the fading is not simultaneous over the whole bright area, but proceeds from one side or other, so that a black shadow seems to sweep across the field and, sometimes, to retreat and advance several times before completely extinguishing the light.

In the case of complete fading of a single patch of light on a dark ground, the coming and going of the image in consciousness seems somewhat arbitrary and lawless, but it is clear that the tendency to fade grows with the duration of fixation, and that it is far greater with relaxed than with accommodated eye, and with a dull than with a bright or very bright image. If, when a small white patch *a* has been fixated for three or four seconds only (the accommodation mechanism being relaxed), a second similar patch *b* is suddenly exposed so that the image of it falls on another part of the same retina, *or on a non-corresponding part of the other retina*, the image *a* merely exhibits some degree of loss of brightness; but if *a* is fixated for about fifteen seconds or more and *b* then suddenly exposed, *a* will usually disappear

[1] Steady fixation is essential, as the slightest wavering of the fixation prevents complete fading or at once restores the image to consciousness if faded. To fixate steadily and at the same time to maintain the accommodation mechanism in a state of complete relaxation requires considerable practice. A method by which anyone may observe this curious phenomenon of complete fading without previous practice in the control of the eye-muscles is the following: Paste a sheet of white paper on a window-pane and fix flat against the paper a sheet of coarsely perforated zinc with circular or hexagonal holes from 1 cm. to 3 cm. in diameter; then fixate with one eye only a point about the centre of the sheet of zinc. The bright circles or hexagons may then be seen to pop in and out of consciousness in a very curious way, some three or four being usually absent from consciousness at any one moment. With relaxed accommodation and a + 4 D glass the effect may be observed still more readily.

from consciousness at once, remain absent several seconds, and then return suddenly, appearing equally bright with *b*.[1]

In the paper on vision[2] I have described numerous instances of this inhibitory effect exerted by one visual image on another, and have pointed out its importance for the theory of vision, endeavouring to show, among other things, how it plays the chief part in all cases of simultaneous visual contrast. Here I am only concerned to show how the phenomena of complete fading and mutual inhibition of visual images lend support to the proposition that consciousness is determined at the synapses.

In the case of complete fading of a single image on a dark ground the light rays continue to effect in the retina the usual chemical changes,[3] and we are therefore compelled to assume that the excitation, set up by these chemical changes in the neurones of the retina, ceases to be propagated through the higher chain of neurones of the cortex in which consciousness is chiefly or wholly determined. And since, when the image returns to consciousness after an absence of one or more seconds, its brightness is not perceptibly less than before its disappearance, we cannot ascribe the failure of the excitation-process to pass through the higher neurones to any diminution of its energy. We are driven rather to suppose that at some point in the conduction-path the normal resistance to the passage of the excitation rises during continued activity until it reaches such a degree as completely to block the path, and that, during the short period of absence of the image from consciousness, this higher path being at rest quickly regains its former state, *i.e.*, the increased resistance rapidly falls again to such a point that the excitation passes through the path once more. Now, in the preceding sections of this paper we have already reviewed the evidence that points unmistakably to the synapses as the chief seats of resistance in any conduction-path and as the seats of the changes that produce variations in the degree of the resistance, and in the light of that

[1] For full descriptions of such cases see Observations x. and xi. in the paper referred to above. *Mind*, vol. x.

[2] *Ibid.*

[3] For proof of this see Observations vi., vii., and viii. *Loc. cit.*

evidence it appears in the highest degree probable that in the case of complete fading of visual images the increase of resistance is determined at the synapses. When, after fixating a patch of light *a* for about fifteen or more seconds, it is made to disappear from consciousness by throwing a similar image *b* on to another part of the same *or the other retina*, this can only be because in some way the rise of the image *b* to consciousness, or better, the passage of the excitation set up by it through the neurones of another part of the visual cortex, tends either to increase the resistance of the synapses of the conduction-path of *a* or to diminish the energy of the excitation-process seeking to pass by that path, which tendency is not powerful enough to prevent the passage of the excitation through the conduction-path of *a* unless the resistance of the synapses of that path has been raised by a previous period of activity of fifteen or more seconds. To discuss the nature of the inhibitory effect would carry me too far away from the subject in hand, and it is to the fatigue effect that I wish to draw particular attention, for on this depends the strongest strand of my argument.

For the occurrence of this increase of resistance of the cortical conduction-paths through fatigue of the synapses we may find abundant evidence, besides that of the kind adduced above, in the phenomena of the struggle of the two visual fields. It is a familiar fact that when we look at a bright white surface, with a red glass before one eye and a blue glass before the other, the surface usually appears alternately red and blue; there is an alternation of the red and the blue images in consciousness, *i.e.*, the blue image of one eye fades completely and the red image alone affects consciousness, until after a short period, that varies in length somewhat under different conditions, the red image fades completely and the blue one alone affects consciousness. For a description of a variety of cases and a discussion of binocular, and especially of monocular, struggle of colours, I may refer to my paper on vision.[1] The point on which I wish to insist here is that when the one coloured

[1] *Loc. cit.*

image disappears from consciousness, the neural events underlying that disappearance seem to be similar to those underlying the simple complete fading of a single visual image, or of one image inhibited by a second image on another part of the same or other retina; that is to say, the excitation ceases to pass through the neurones of the visual cortex, and the factor that determines the regular alternation of the two images is the rapidly oncoming and rapidly disappearing fatigue of the synapses of the cortex.

In support of this view we have the following facts:—(1) A very similar alternation in consciousness, *i.e.*, alternate predominance of an image and inhibition of it by another image, may occur in the case of two after-images formed on adjacent areas of one retina;[1] (2) if, when a red field is presented to one eye and a blue field to the corresponding area of the other eye, one eye be closed or covered for a brief period—one second will suffice—the colour presented to that eye always predominates over and inhibits the colour presented to the other eye as soon as the eye is uncovered, *i.e.*, the rested tract predominates over the relatively fatigued tract, even if the period of rest be not more than one second; (3) if by any one of several devices, as, for example, slight movements of objects in the field (the drawing of a hair across the field a little before the eye will suffice), the predominance of one field, say the red field, be prolonged, the blue field of the other eye tends more and more strongly to assert itself until, no matter how vigorous the movements in the red field, the blue predominates and the red field disappears from consciousness; all such devices merely direct attention to the one field, *i.e.*, they cause the excitation of the one cortical conduction-path to be re-enforced by the activity of higher levels, and we see that as the excitation continues to pass through this path the resistance of the path increases until, in spite of such re-enforcement, it yields to the inhibitory influence of the other rested tract; (4) if, accommodation being relaxed, a white field be presented to the right eye and a much less bright white or gray field to the corresponding area of the

[1] Observation xiii. *Loc. cit.*

left eye, and if the left eye be covered over for some ten to fifteen seconds, while the right eye continues to fixate the brighter field, then on uncovering the left eye the bright image of the right eye yields to the much less bright image of the left eye, and disappears from consciousness for some seconds. I hold this last observation to be especially important, because it shows that the relative degree of fatigue of the conduction-paths is of more importance than the relative brightness of the two images in determining the predominance of one or other of them.

The evidence of fatigue in the cortical conduction-paths thus afforded by the study of visual sensation corresponds in a striking manner with that yielded by Levy's experiments in direct stimulation of the cortex. Both kinds of evidence point to the occurrence in the cortex of a peculiar form of fatigue not assignable to the neurones. The sudden fall of the contraction-curve in Levy's experiments seems to correspond to the disappearance of the visual image from consciousness; the former occurs after continuous faradic stimulation for a period of from eight to thirty seconds, while the visual image shows no pronounced tendency to fade until after it has been present to consciousness for about ten or fifteen seconds. In both cases the failure of the cortical conduction-paths occurs earlier with a weak than with a strong stimulation. In both cases the fatigue-effect is very rapidly abolished during rest, and in both cases there is evidence that the energy of the excitation process is not diminished, or at least not diminished in such a degree as would account for its failure to pass through the cortical conduction-path. These two kinds of evidence, then, taken together and in conjunction with the abundant evidence from other sources pointing to the synapses as the chief seats of resistance in every conduction-path, a resistance variable in degree, enable us to assert with some confidence that the synapses of the cerebral cortex transmit the excitation from neurone to neurone by a process that is subject to a peculiar form of fatigue characterised by its rapid onset and rapid disappearance, and that this process, the material basis of which is presumably the intercellular substance, is

far more readily fatigued than the processes of any other part of the conduction-paths. Now the psycho-physical processes must be of a very delicate and complex nature, in fact the most complex and delicately adjusted processes of the animal body, and for each primary sensation-quality at least there must be a perfectly specific psycho-physical process, probably in part at least of the nature of a chemical change. *Such a highly specialised process is just such as we should expect to be very readily fatigued, and it is therefore in the highest degree probable that the parts of a cortical conduction-path in which the psycho-physical processes occur are the parts most liable to fatigue. And we have good reason to believe that those most readily fatigued parts are the synapses.*

I will now bring together certain facts which indicate that the psycho-physical processes underlying our subjectively continuous psychical processes are discontinuous, *i.e.*, that they consist in the repetition at very brief intervals of a process that runs its course in a minute fraction of a second, the process of discharge of the afferent into the efferent neurone across the intercellular substance at the synapsis.

We know that series of stimuli applied at sufficiently short intervals to the terminations of the sensory nerve in the sense-organ will give rise to continuous sensations. Thus a regular series of air-waves falling upon the ear at a greater rate than about 16 per second gives rise to a continuous sensation of tone.[1] Touches or induction shocks repeated at the same spot of the skin may give rise to sensation of continuous contact when repeated about 36 times per second. In regard to visual stimuli the reports of different experimenters are not in agreement. Exner, by an ingenious experimental procedure and a somewhat questionable process of inference,[2] arrived at the conclusion that a visual sensation does not reach its maximum intensity until a considerable interval after light reaches the retina,

[1] If we may suppose that each phase of the complete wave acts on the nerve as a stimulus, the number of stimuli per second that gives continuous sensation will be about 32.

[2] Described by Helmholtz. *Physiol. Optik.*, p. 514.

in some cases nearly a quarter of a second; but the phenomenon known as Charpentier's bands seems to prove that a visual sensation may reach its maximum and decline again within one-sixtieth of a second after its inception. Exner gives about 44 σ as the interval that must separate two stimuli to the same retinal area if they are to be felt as two.[1]

Other observers, working with rapid alternations of light and darkness, generally with rotating sectors, have found that the rate or alternation at which fusion to continuous sensation occurs, varies with the colour and brightness of the light. Burch [2] has, however, pointed out that the duration of the intervals of darkness is of primary importance. Now Haycraft,[3] by a procedure that gave equal intervals of light and darkness, found that fusion occurred with the brightest light used at 19 alternations per second, which means a duration of the dark intervals of one thirty-eighth sec. (26 σ). This interval is just that at which successive touches may cease to give a discontinuous sensation (Exner and Preyer).

More important than these observations, which show that successive stimuli give rise to a continuous sensation when sufficiently rapidly repeated, are certain observations which show that a continuous stimulus may give rise to a discontinuous sensation, to a series of pulses of consciousness. Charpentier first observed that when a bright white surface is carried at a moderate speed across a dark field of view, its leading edge appears as a narrow band, that is brighter than the rest of the surface and is followed by an equally narrow dark band, and under favourable conditions he was able to observe as many as four or five alternating light and dark bands all about the same width, a width equal to the space through which the leading edge moves in about one-sixtieth second.[4] Shelford Bidwell confirms this observation and notes also that, when a single narrow band of light is carried across a dark field in a similar

[1] Hermann's *Handbuch d. Physiol.* Bd. II.
[2] *Journ. of Phys.*, vol. xxi. "On Colour-Vision with Intermittent Light."
[3] *Journ. of Phys.*, vol. xxi. "Luminosity and Photometry."
[4] *Compt. Rend.*, 1887.

manner, it appears to him as four or five successive images separated from one another by narrow bands of darkness.[1]

As to the appearances that are caused by a single brief exposure of the retina to a bright patch of light, and as to the phenomenon usually called the "recurrent image," the reports of different observers are strangely at variance and the whole subject is still in a very unsatisfactory state. The only fact on which all are agreed, I think, is that there occur rapid variations in the brightness of the sensation. My own observations agree most nearly with that one of Bidwell's mentioned above. This is not the place to describe them in detail or to attempt to correlate them with the various descriptions given by different observers. But since they afford evidence of the discontinuous character of the psycho-physical processes underlying continuous sensations they must be briefly mentioned. I have used various arrangements for securing the brief exposure of a patch of light, including one that ensures absolutely simultaneous appearance and disappearance of the light over the whole of the illuminated area. I find, however, that the slightest noise, such as is made by a rapid photographic shutter, renders quite impossible (for me, at least) the satisfactory observation of the brief visual image. I will describe therefore a series made by the following very simple method, noting that the other methods yield similar results, though less distinctly.

I obtained an oculist's trial-frame surrounded by opaque silk somewhat in the fashion of a motorist's goggles; the left aperture was covered in completely, the right one fitted with a metal disc with a circular hole 2 mm. in diameter at its centre, so that all light, save what reaches the right eye through this hole, is excluded. Wearing this arrangement I sit opposite to, and about three yards distant from, a vertical slit in the window-shutter of the dark-room, about 3 cm. in width and 10 cm. in length. In my right hand I hold a large sheet of black cardboard fashioned somewhat like a large Japanese fan, with wooden handle below, and in

[1] "Curiosities of Light and Sight," p. 191. London 1899; and *Proc. Roy. Soc.*, 1894.

its upper part a vertical slit about 5 cm. in length and 1 cm. in breadth. By a small movement of the wrist this slit can then be carried rapidly across before the right eye and when the eye is directed towards the bright patch of light formed by the aperture in the window shutter, that is exposed to the eye for a fraction of a second and then immediately extinguished. On a bright winter's day, when the sky is a bright white surface, a sheet of coloured glass is put over the aperture in the shutter so that the eye, looking directly at the sky through the aperture, receives the image of a little slip of bright colour. When in this way a bright coloured image is allowed to fall on the retina for a moment, the character of the image is similar for all three colours used by me, namely, red, green and blue, and for a considerable range of duration of the exposure, from about one-fifth second downwards. The effect falls distinctly into two stages; the first stage consists in a pulsating or quivering image of the same colour as the glass used, declining rapidly in brightness, each pulse being less bright than its predecessor, until, after a period that increases with the brightness of the light from a small fraction of a second to nearly one and a half seconds, this first stage ceases and is replaced by a steady dull white after-image that dies away in the course of ten or more seconds. The pulses of colour are not separated by distinct intervals of darkness, rather each seems to begin before its predecessor has completely disappeared and the effect resembles the "coarse flicker" of of rotating sectors. The rate of the pulsation is slower in the image of a dull than of a brighter light, and it seems to decline with the brightness, so that, towards the end of this pulsating stage of the image of a light of moderate brightness the pulses can just be counted (which means that they are coming at about the rate of ten per second). At a time when the sun is opposite the window and shining through a thin white veil of haze in an otherwise clear sky I can, by repeating the observation with my eye in a series of positions such that it receives the light from parts of the sky successively nearer to the sun's disc, observe the effects of light of increasing brightness. With each of

the three colours the effect is then similar ; with increase of brightness of the light the duration of the pulsating image increases and the pulsations become more rapid ; with light from the sky close to the sun's disc the effect resembles the "fine flicker" of rotating sectors ; and by choosing positions nearer and nearer to the direct ray of the sun it may be observed that the pulsating or quivering becomes so rapid as only to be noticeable when close attention is given to it ; and then, finally, when the direct rays of the sun fall on the eye, the bright image persists for some seconds as a continuous steady sensation, *i.e.*, without any perceptible quivering.

This series of observations suggests very strongly that the momentary action of the bright light on the retina in some way overcharges the sensory neurones, so that they discharge themselves through the conduction-paths concerned by a rapid series of discharges of diminishing energy analogous to the oscillatory discharge of a Leyden jar, each discharge causing or constituting a psycho-physical process, or, as it may be called, a psycho-physical flash. And the transition from the slowly pulsating image through images of more and more rapid pulsation to an image that is perfectly and smoothly continuous, suggests that in the case of this last also the psycho-physical process underlying it is a series of such flashes very rapidly repeated, rather than a continuous process. If we accept this hypothesis provisionally it will lend itself well to the explanation of the effects of rapidly repeated stimulation of sensory nerves and of Charpentier's phenomenon, briefly reviewed above. We must suppose each effective stimulus applied to a sensory nerve, to give rise to at least one discharge, one psycho-physical flash. Thus in the case of sight and touch it would seem that when the stimulus, and therefore the psycho-physical flash, is repeated at shorter intervals than 26 σ, the resulting sensation is continuous in character, but discontinuous when the psycho-physical flash is repeated at greater intervals. In the case of Charpentier's phenomenon we must suppose that, under the conditions of the experiment, the moderately bright light is unable to cause the

repetition of the psycho-physical flash at a greater rate than thirty times per second, *i.e.*, at intervals of about 33 σ, in the first moments after it begins to act on any given part of the retina; that is to say, each newly-stimulated part of the cerebro-retinal apparatus, being under the inhibitory influence of the image of the white surface formed on adjacent parts of the apparatus, exhibits a certain inertia, so that for a brief period of time the psycho-physical flash is repeated at greater intervals than 26 σ and the accompanying pulses of consciousness are felt individually. Bidwell's multiple images of the moving streak of light would represent again the successive psycho-physical flashes repeated at a greater interval than 26 σ.

The Psychological Evidence.

In this section I propose to recapitulate as shortly as possible the argument presented in support of my proposition in the paper before referred to.[1]

If, taking a general view of mental processes and directing our attention more especially to those involving clear or focal consciousness, we ask—What is the essential characteristic of mental activity? we find that it consists in the process of establishing in the mind relations between one thing and another. This process in its best-defined form is what is known as apperception, "the process by which a mental system appropriates a new element or otherwise receives a fresh determination."[2] The whole mind is shown to be organised into mental systems of very different degrees of complexity, corresponding to more or less complex objects;[3] each mental system is gradually built up by a series of apperceptive processes, each such process consisting in the presentation of some one aspect or feature of the whole object through some sense-organ, and the bringing of this feature into mental relation with various other aspects

[1] *Mind*, vol. vii.

[2] G. F. Stout. "Analytic Psychology." The modern doctrine of noetic synthesis and the growth of mental systems by successive processes of apperception is admirably set forth in this work.

[3] The word "object" is used here in its widest sense.

and features previously apperceived and incorporated into the mental system. "In almost every moment of waking life an apperceptive process is taking place; whenever an object is attended to the presentation of it is apperceived."[1] Mental activity, then, consists essentially in the perpetual succession of apperceptive processes, and the essence of apperception is the appropriation of the relatively novel presentation by the mental system built up by previous apperceptions, or, more shortly, is the bringing of the new into relation with the old, the product of past mental process. At each apperception of any given presentation of an object the appropriation of it by the mental system is more ready and more complete, while the consciousness excited by this aspect of the object becomes less and less vivid, until finally, when the appropriation of it by the mental system becomes complete, it is implicitly apprehended, or, in terms of conation, the stimulus supplied by this aspect of the object is responded to automatically, while some other aspect occupies the focus of consciousness.

When we attempt to translate all this into terms of neural process, our account must run in some such way as follows:—The mass of neurones constituting the nervous system is organised into functional groups and sub-groups, the members of each such group being intimately united together so that the excitement of any member of the group is shared by all the rest; such a group is the physiological counterpart of a mental system, or better, is the mental system in its physiological aspect. That an object is familiar to us means that, by successive presentations of its various aspects, the group of neurones directly excited by the presentation of any one aspect, has become intimately functionally related with the groups directly excited by the presentations of other aspects of the object. When we attend to any one aspect or feature we are apperceiving that feature, that is, we are relating it to other features, and this means in turn that the group of neurones directly excited by it is being brought into functional relation with those

[1] Stout. *Op. cit.*, vol. ii., p. 113.

groups which have been previously organised by a similar process into the mental system, or complex group of neurones, corresponding to the total object. The physiological process constantly accompanying or underlying the focal consciousness of states of mental activity is then a process by which one group of neurones is brought into more intimate functional relation with another, generally a more complex, group of neurones. Now so far as we can at all conjecture, such an association of two groups of neurones means a change at the synapses which constitute the physiological boundary and points of contact between the neurones of the one group and those of the other (*i.e.*, physiological contact, not necessarily physical contact); a change of such a nature that thereafter any excitation can spread more readily across the synapses from one group to the other. Such a change, such a diminution of the resistance at synapses, seems to be the normal result of the passage of the excitation process across them.[1] Focal consciousness seems, then, to have as a constant physiological accompaniment the process by which the excitement of one group of neurones overcomes the resistance of a set of synapses and spreads across them to another group; and as these synapses become organised —*i.e.*, as their resistance becomes reduced more and more by repetition of the discharges across them—the consciousness accompanying the process becomes less and less clear and less intense until, when the synapses have become fully organised (*i.e.*, have their resistance reduced to a minimum), it is of a very obscure nature, forming at most only a part of what James[2] has called "the fringe of thought"; for the two groups of neurones have then become one functional group, one mental system, and a presentation, that formerly directly excited the one group only, is now implicitly apprehended and automatically reacted to, the excitation-process set up by it spreads through the whole mental system

[1] Of this we have fairly direct evidence in the shortening of reaction times with repetition; but beyond this, it is no exaggeration to say that without this assumption it is impossible to begin to attempt to correlate psychic and neural functions or to form the vaguest conception of the physiological change underlying association and constituting the physical basis of memory.

[2] "Principles of Psychology."

without having to overcome the resistance of unorganised synapses, and without arousing focal consciousness.

The spinal cord and lower parts of the brain seem to consist wholly, with the exception of course of the long tracts of fibres merely passing through them, of completely organised groups of neurones, elementary mental systems, built up long ago in the history of the race by processes involving focal consciousness, and now merely the fixed reflex conduction-paths. While in the cortex, at the top of the whole nervous system, are neurones and groups of neurones not yet built up into mental systems, together with immensely complex mental systems capable of apperceiving a great variety of presentations, *i.e.*, of being brought in successive acts of attention into relation with various other groups of neurones, by the passage of the excitation-process across the synapses forming the physiological barriers between the groups.

The one physiological process directly correlated with focal consciousness is, then, this passage of the excitation across relatively unorganised synapses, and this would therefore seem to be, according to the terms of our definition, the psycho-physical process.

The same conclusion may be reached by looking at the facts from another point of view. The only objective evidence of the occurrence of consciousness is afforded by proof of retention by the nervous system. Where there is evidence of retention, of a functional change due to the activity of the nervous system, there we may conclude that consciousness accompanied that activity. Where any nervous activity proceeds automatically, *i.e.*, without consciousness, there we never find evidence of retention. Professor Bain[1] has pointed out this constant association of consciousness with those forms of neutral activity that lead to retention, and the same generalisation has been vigorously though somewhat grotesquely stated by Loeb in a recent work. He writes[2] that "so-called psychic phenomena appear, invariably, as a function of an elementary process, namely, the activity

[1] *Mind*, N.S., vol. iii.
[2] "Comparative Physiology of the Brain and Psychology." London, 1901, pp. 213-214.
[3] *Mind*, vol. vii.

of the associative memory"; and again, "I think it can be shown that what the metaphysician calls consciousness are phenomena determined by the mechanism of associative memory." In the essay before referred to I have put the argument in the reverse and, as it seems to me, the correct order, namely, that what the plain man calls consciousness determines those effects which Prof. Loeb attributes to the activity of a metaphysical entity, namely, the associative memory. But, whatever way the truth may lie as to the causal relation, the important point for the purpose in hand is that the two things are constantly together; wherever there is memory or other evidence of retention, consciousness accompanied, and probably was one of, the conditioning factors of that retention.

Now retention means the persistence of some physical change in the conduction-paths. And the persistent change which is the physical basis of memory or retention can only be conceived as consisting in such a union of the units composing a conduction-path that they shall in future tend to be simultaneously active, and this must mean an increased intimacy of connection between neurones, the result of organisation of the synapses through which they act upon one another. Similarly, the physical basis of that form of retention known as the association of two ideas by contiguity in time can only be conceived as an organisation of, a diminution in resistance of, the synapses through which the two conduction-paths, whose activity is the physical basis of the two ideas, can act upon one another. Such organisation of synapses is, as we have seen reason to believe, the result of the passage of the excitation process across them, which passage across unorganised synapses is then the only physiological process known to be constantly accompanied by consciousness, and therefore in all probability constitutes or immediately determines the psycho-physical process.

The Application of the Conclusion arrived at.

There is, then, a considerable mass of evidence, histological, physiological and psychological, pointing to the

synapses, more especially those of the cerebral cortex, as the seats of the psycho-physical processes, while, so far as I can discover, there is no reason of any kind whatever to suppose that the cell-bodies are the seats of those processes, as has been so commonly assumed. No one piece of this evidence, taken alone, is of a kind to force upon us the acceptance of this view, and from the nature of the case we cannot hope that any such direct and unmistakable evidence will ever be forthcoming. In this final section I wish to show that the conclusion arrived at is not one of theoretical interest only, but is such as to lead us on to new and fruitful conceptions as to the workings of the nervous system.

In a recently published pamphlet [1] von Kries has pointed out that, in spite of the immense progress made of late years by our knowledge of the structure of the nervous system, our knowledge of its functions seems by comparison to have remained almost stationary, owing to the lack of new conceptions distinctly physiological, not merely anatomical, in character. Von Kries believes that this is due to an unwarrantable acceptance of what he calls the conduction-hypothesis (Leitungsprinzip), the hypothesis that all nervous activity consists in the propagation of some state or process of excitation from part to part, such as we observe in the peripheral nerves, the nature or quality of the process varying according to the properties of the conduction-paths. He argues that this conduction-hypothesis has proved itself barren and that it is incapable of enabling us to describe even in vaguest outline the more complex functions of the brain, and he attempts therefore to give new vitality and increased definiteness to the hypothesis of the individual functioning of the ganglion-cells, the view that each cell is a psychic unit capable of determining within itself a more or less complex state of consciousness.

It does not seem to me that v. Kries makes out a sound case against the conduction-hypothesis, and the alternative that he proposes would seem to stand towards neurology in a relation similar to that of the doctrine of vitalism towards

[1] *Ueber die materiellen Grundlagen der Bewusstseins-Erscheinungen*, Mohr, Tübingen, 1901.

physiology in general, and to be therefore rejected so long as there is any hope of progress along other lines. But it must be admitted, I think, that, if the conduction-hypothesis is to remain our fundamental conception of nervous process, it sadly needs to be refined and fertilised by supplementary hypotheses. Towards the formation of such a hypothesis, a distinctly physiological conception, I hope to contribute my mite by drawing in rough outline a scheme that shall bring under one point of view the various facts dealt with above, together with the conclusion arrived at as to the functions of the synapses. And first, I would venture to urge that physiologists ought not to hold themselves slavishly dependent on the physicists, but that they must press on towards the solution of their own problems, building up for themselves such conceptions as shall enable them best to describe the peculiar processes that they have to study. Nor, in forming such conceptions, should they allow themselves to be hampered by a too great desire for formal consistency, but should follow the example of the physicists in this respect. The most wonderful achievement of our time is perhaps the command over, and insight into, electrical processes acquired by physicists, yet it would seem to be still impossible to describe the phenomena consistently in terms of any one of the several hypotheses as to the nature of electricity; again, the most fruitful hypothesis ever developed is perhaps that of an ethereal medium to which an impossible and inconceivable combination of properties is assigned by physicists.

We have seen above that Schäfer's suggestion of a latent period of excitation of each neurone cannot be satisfactorily applied to the explanation of the "block" between neurones and of the rhythmic discharge of efferent impulses. Another suggestion recently put forward by Professor Richet[1] must be briefly mentioned. Richet seeks to explain the phenomena of the retardation of the excitation-process in the central nervous system and the rhythmic efferent discharge by attributing to every nerve-cell a refractory period of nearly $100\,\sigma$ following the passage of each impulse through it. He

[1] Address to Brit. Assoc., Dover, 1898, reported in *Nature*.

regards neural activity as consisting in a series of diphasic waves, of which the negative phase constitutes the refractory period of the nerve-cell. I do not know that there is any one fact that will suffice to disprove this view outright, but we may note in the first place that Richet offers us, as proof of the wave-character of the excitation-process, merely the fact that it can be diagrammatically represented as a wave. In a very similar manner we may represent a man's appetite for food and drink, or the war-like feeling of the country, and many other things, as diphasic waves of which the negative phase corresponds to a refractory period; but to do this is merely to speak in terms of analogy, and by no means proves that these happenings consist of wave-movement in a physical medium. Further, we know that a nerve-fibre will respond to each one of a rapid series of stimuli, and that if it has any refractory period its duration must be less than 1 σ,[1] while Charpentier's phenomenon seems to prove that the psycho-physical process may be repeated much more frequently than ten times per second, at intervals certainly not greater than 30 σ. This view, too, would seem to offer little promise of leading us to an effective method of describing the workings of the nervous system, and to be in this respect much inferior to the one that must now be dealt with.

Many considerations indicate that, in the workings of the nervous system, an important part is played by the charging of the individual neurones with an undefined something, and that a certain potential of charge must be given to any neurone before it can communicate its excitement to others, *i.e.*, before the charge can overcome the resistance offered by the synapses.

Exner recognises the necessity of adopting some such conception when he writes: "The reflex contraction in reply to a momentary stimulus has always a course very different to that of a contraction evoked by direct stimulation of the centrifugal nerve. It lasts longer, and may prolong itself to a tetanus. That indicates that somewhere in the

[1] *Cf.* F. Gotch, Art. "Nerve," p. 475 in Schäfer's "Text-book of Physiology."

centre (centrum), let us suppose in the end-twigs or in the ganglion-cells, a sort of charging (Ladung) takes place under the influence of the stimulus, and that this charge of the centre discharges itself through the outflow of the excitation to the muscle."[1] And throughout this work this idea of a "Ladung" of centres is frequently introduced. Bechterew[2] has given expression to an hypothesis of this kind, suggesting very roughly an analogy between the chain of neurones and a chain of Leyden jars. In an important and interesting essay Goldscheider[3] recognises the need of some such conception, and propounds the doctrine of the "neurone-threshold," the importance of which for therapeutics and pathology he insists upon. He recognises "that the principal conduction-resistance is offered by the passage (Uebertritt) of the excitation from neurone to neurone" (p. 5), and that the varying degrees of this resistance account for varying periods of lost time in the same and in different conduction-paths. "We must attribute a certain inertia (Trägheit) to the process that intervenes between the neurones (dem zwischen den Neuronen sich abspielenden Vorgange); otherwise we cannot understand the isolated conduction through the labyrinth of neurones in the gray matter" (p. 9). He admits the possibility that this resistance may be conditioned by an inter-cellular substance, but he thinks it improbable, and he attributes both the facilitation and organisation of conduction-paths (Exner's "Bahnung" and "Ausschleifung") to the neurones. While recognising the importance of this doctrine of the neurone-threshold and the enlightenment that it brings us, I venture to think that we shall receive still greater enlightenment if, accepting the indications afforded by all the different kinds of evidence adduced in the preceding sections of this paper, we assign the principal resistance in all conduction-paths to the inter-cellular substance at the synapses and so convert the doctrine of neurone-thresholds into one of synapsis-thresholds. The adoption of the threshold hypothesis in this form

[1] *Entwurf zu einer physiologischen Erklärung der psychischen Erscheinungen*, p. 49. Wien, 1894.
[2] *Neurolog. Centralblatt*, 1896.
[3] *Die Bedeutung der Reize im Lichte der Neuron-Lehre.* Leipzig, 1898.

brings with it at once a great and obvious advantage. It introduces a great refinement into our conception of the mechanism that determines the course of any excitation in passing through the labyrinth of paths from afferent to efferent nerves. In place of regarding each neurone as having a certain threshold common to all its dendrites, each pair of fibrils that meet in a synapsis will be regarded as separated by an inter-cellular substance whose degree of resistance or threshold has been determined by the history of that synapsis in the individual and the species, and our conception of the mechanism that determines the course of any excitation will then be refined in proportion as synapses are more numerous than neurones.

The idea of a certain charging of neurones has then been entertained by authors of high authority, but I have not been able to discover that anything has been said as to the nature of that something with which the neurones are supposed to be charged, although an effective working conception of its nature must be of the highest importance if there be any truth in this view. I think that for the present it may be best conceived as a fluid, and I propose that this fluid shall be called "neurin."[1] It might of course be called the nervous fluid, or nervous energy, or "animal-spirits," or a very subtle ether, but the name I suggest is preferable because it implies nothing beyond the fact that the thing named has to do with nerves. I have little doubt that at some future date it will be regarded as some form of motion of some species of ether, or of some still more remote hypothetical medium. But just as the fluid-theory of heat, the two-fluid-theory of electricity and the corpuscular theory of light furnished probably the most useful working conceptions for the sciences of heat, electricity and light at certain stages of their development, so neurin may, I think, be most usefully conceived as a fluid in the present state of neurology, and I think it would be unwise to attempt to regard it as a variety of any one of the forms of energy known outside the animal body, although it is easy to

[1] I am indebted for the suggestion of this word, the adjective form of *νευρον*, to my friend Prof. G. C. Moore Smith.

discover points of resemblance to both electricity and magnetism.[1] As Barker writes: "It is by no means impossible that in the nervous system forms of energy are concerned which do not exist outside the animal body and which yet remain to be recognised and studied;"[2] and we may perhaps go even further than this and say that the existence of some such form or forms of energy may be assumed with a very high degree of probability; for since, so far as we know, consciousness is only associated with transformation of energy within the living animal body, the forms of energy concerned must be of a highly special and peculiar nature.

I will therefore sketch in rough outline a scheme of the part that the fluid neurin seems to play in the workings of the nervous system. In virtue of its normal vital activity every neurone continually produces neurin in small quantity; and the neurones connected with the sense-organs and surface of the body generally are almost perpetually played upon by feeble stimuli that excite them to the production of neurin in rather larger quantities. Neurin tends always to flow from a place of high potential to places of lower potential. The neurin so produced therefore tends continually to flow from afferent to efferent neurones, in virtue of the higher potential of the sensory neurones and of the valve-like nature of the synapses, passing across the synapses by a sort of leakage, and so escaping by the efferent nerves into the muscles as a continuous gentle stream, maintaining that state of continued gentle contraction of the muscles which we call their tone.[3] It is possible that the slow leakage across synapses is the basis of that obscure affection of

[1] I do not mean to imply that the numerous attempts that have been and are being made to exhibit the nervous impulse as an electrical phenomena should not be regarded with the fullest approval. I mean that we ought not to wait for the final issue of such attempts before endeavouring to define more fully the nature of neural processes.

[2] "The Nervous System and its Constituent Neurones," p. 249.

[3] In this connection it should be remembered that in spite of the oft-repeated statement to the contrary effect, a continuous current of constant strength is capable of bringing about a continued contraction when thrown into a muscle-nerve preparation under suitable conditions. *Cf.* Biedermann "Beiträge z. Kenntnis de Reflex Function des Rückenmarkes," p. 410, *Pflüger's Arch.*, Bd. 80.

consciousness which has been called the "general sensibility" and forms the groundwork of the psychological self.

When any adequate stimulus falls upon a sense-organ the sensory neurones affected by it are excited to the production of much larger quantities of neurin per unit of time. Let us take the case of a ray of light falling upon the retina and there setting free a substance that chemically stimulates the nerve-endings, and let us call the neurones forming a conduction-path leading from the sense-organ to any motor neurone, *a*, *b*, *c*, *d*, &c., each one, as *b*, being efferent to its predecessor in the chain *a* and afferent to its successor *c*. Then *a*, continuously stimulated by the exciting substance in the retina, produces neurin far more rapidly than it can escape by leakage into *b*, so that in a small fraction of a second the potential of the charge in *a* will rise to the level of the threshold of the synapsis *a*—*b*. A sudden discharge of neurin then takes place across the synapsis from *a* to *b*, and its sudden arrival in *b* acts upon it as a stimulus to the rapid production of more neurin, so that *b* then has a double charge of neurin and therefore rapidly discharges into *c*; and so the process of discharge and stimulation of afferent into efferent neurone is repeated, until the last of the chain discharges itself into the muscle and brings about contraction. To go back now to the sensory neurone *a*; as it discharges, the exciting substance in the retina continues to stimulate it to rapid production of neurin, and therefore, in a very short time after the first discharge, the potential of its charge rises again to discharging point, and so once more the whole chain of neurones is charged, stimulated and discharged in turn, and a second impulse flows out to the muscle, and so on again and again. It is necessary to assume that the neurones at the efferent end of the chain have a larger capacity for neurin than those of the afferent, or of the central parts, so that, while the latter discharge many times a second (more than thirty-six times per second as we have seen in case of a continuous visual sensation), the large motor neurones of large capacity have their potential raised to discharging point less rapidly and therefore discharge at a

slower rate—about 10-20 times per second, according to the intensity of stimulus and other conditions. And the conduction-path excited is probably never so simple as suggested above, there is probably always some spreading out of the path, so that for each sensory neurone stimulated several neurones towards the efferent side are affected, and the more intense the stimulus the greater the spread of conduction-path involved.[1] For with the minimal effective stimulus neurone *a* will only discharge by the synapsis of lowest threshold into perhaps one *b* neurone only, while with stronger stimulus the potential of *a* may be raised so rapidly and to so high a point that discharges take place by synapses of higher threshold into other *b* neurones. And this may be repeated in the case of each of the *b* neurones and of the neurones of the succeeding levels; so that, as we know, if the stimulus be a very strong one and the general state one of high excitability, almost all the muscles of the body may be thrown into activity.

This view of the charging and discharging of neurones not only brings under one point of view the loss of time at the synapses and the rhythmic discharge in the efferent nerves, but is supported and, we may almost say, necessitated by an array of facts that find a ready explanation through it. Of these the chief are certain peculiarities of the reflex response, the phenomena of summation of stimuli and of facilitation (Bahnung), and certain facts which prove the existence of a state of residual excitement or rather the persistence of residual charges of neurin after the passage of an excitation.

"There is a distinct relation between the intensity of the external stimulus and the latent time of spinal reaction, the latter becoming much shorter as the former is increased,"[2] and the lost time may even become inappreciably short.[3] The more powerful stimulus leads to a more rapid production of neurin, so that the potential of the sensory neurones sooner passes the synapsis-threshold.

[1] Probably there is usually a spreading out in the middle parts of a conduction-path, and some reconcentration on the motor side.

[2] Sherrington. "The Spinal Cord," Schäfer's "Text-book," p. 829.

[3] Rosenthal, quoted by Exner. *Entwurf*, &c., p. 55.

"A single induction shock, the discharge of a Leyden jar, a single prick, &c., can excite a reflex action. Such brief 'single' stimuli must, however, in order to produce reaction, be violent, so violent as to injure the sensifacient tissue too much for repetition. Even when thus strong they are less efficient than less severe stimuli applied not singly but repeatedly."[1] The effect of a stimulus is to hasten during its incidence the normal slow production of neurin, rather than to give rise to an explosive setting free of energy, hence the importance of the duration of the stimulus.

"The reflex arc, as judged by the contraction of its muscles, behaves somewhat like cardiac muscle in responding its approximate best or not at all. In other words, internal conditions affect the amount of response much more than does mere intensity of external stimulus."[2] In the simple conduction-path of a spinal reflex the resistances of the well-organised synapses are a comparatively fixed quantity; hence the amount of the charge and discharge of neurin is approximately constant.

"Stimuli far too weak to singly evoke reflex action, are easily able to do so if repeated; with separate electrical or mechanical stimuli the reflex tends to appear after a given number of stimuli have been applied, although the time intervals between the stimuli may be varied within wide limits—*e.g.*, ranging between ·05 sec. and ·4 sec."[3] Each stimulus causes the generation of a quantity of neurin in the sensory neurone too small to raise its potential to the synapsis-threshold, but, when the stimuli are repeated at such a rate that each fresh quantity is formed before any considerable fraction of the preceding charge has leaked away, the charges accumulate until the potential reaches the synapsis-threshold. In such cases the failure to discharge may perhaps occur, not in the sensory neurones, but in the efferent neurones of large capacity. A similar summation of stimuli is illustrated by the cough that follows prolonged slight irritation of the throat (Exner). Urbantschitsch[4] has

[1] Sherrington. *Loc. cit.*, p. 828.
[2] Sherrington. *Loc. cit.*, p. 829.
[3] Sherrington. *Loc. cit.*, p. 828, quoting James Ward.
[4] Quoted by Exner. *Entwurf. z. einer physiol. Erklärung, &c.*

shown that the note of a very gently-sounding tuning fork may fail to be heard until it has fallen on the ear for one or more seconds. This would seem to be a case of summation of stimuli entirely comparable to the foregoing cases.

Exner has shown[1] that if a spot of skin and a spot on the motor cortex be found, stimulation of which leads to contraction of the same muscle in both cases, then weak stimuli applied to these two spots re-enforce one another whether they be separately sub-minimal or not, and whether they be simultaneous or successive at any interval less than three seconds. This is the typical instance of what Exner has called "Bahnung," a word translated by Sherrington as "facilitation." Exner himself regards summation of stimuli as but a special case of "Bahnung," and the explanation in terms of neurin would seem to be entirely similar; in the case described the two conduction-paths, that from the spot on the skin and that from the cortex, converge at some point in their course to become one, and in this part, whose neurones are common to both paths, accumulation of charges of neurin takes place, whether through simultaneous charging from both directions or through summation of successive charges.

Exner[2] has pointed out that the preparation for a reaction, effected by expectant attention, consists in a charging of the conduction-path concerned, and is a process essentially similar to the facilitation of the paths in the last-mentioned experiment. We must suppose that, in response to the premonitory signal, the neurones of the path are charged with neurin almost up to the synapsis-thresholds, so that, when the expected stimulus falls on the sensory neurone, it has to increase the charge of neurin in it but very slightly to lead to its discharge, and so to the successive discharge of the whole chain of neurones constituting the conduction-path to the muscles. As Exner has pointed out, there is no further act of will involved in this response to the stimulus; the whole process takes place automatically, and may be brought about by any sudden sensory stimulus, and, what is particularly significant, it

[1] *Op. cit.*, p. 78. [2] *Op. cit.*, pp. 158 and 131.

may occur after the subject has ceased to intend to react.

The phenomena of facilitation and the summation of stimuli afford evidence of the persistence in neurones of a charge of neurin for some seconds, and there is other evidence pointing to the persistence of a residual charge after the discharge of neurones. In the case of the cord this is indicated by experiments which prove that the production of a reflex is rendered easier by an immediately preceding activity of the conduction-path concerned.[1] Similar evidence in the case of neurones of the brain is afforded by certain psychological observations. In an important paper recently published,[2] G. E. Müller and Pilzecker have shown that an image (they dealt chiefly with nonsense-syllables) that has occupied consciousness tends to rise again to consciousness spontaneously, as it were, *i.e.*, without any assignable reproducing association, a tendency to which they give the name "Perseverations-tendenz"; and they show that this tendency is strong at first but declines rapidly, so that after a few minutes it but rarely makes itself felt. And they have further shown that during a similar period, following the succession of two images in consciousness, the association between those two images continues to increase in strength. My suggestion is, that there remains in the group of central neurones concerned in the production of an image a residual charge of neurin, so that their potential remains not far below the discharging point, and a very slight stimulus will cause their discharge, and so the revival of the image in consciousness; and that the gradual escape of this residual charge, by leakage across synapses, is the cause of the continued diminution of resistance of the synapses, *i.e.*, of increasing fixity of the association. The same authors show that the time needed for the reproduction of an image by an associated image is much shorter immediately after the image has passed from consciousness than a few minutes later, while the certainty of

[1] Sherrington. *Loc. cit.*, several experiments, p. 842.

[2] Müller and Pilzecker. "Experimentelle Beiträge zur Lehre vom Gedächtniss," *Zeitsch. für Psychologie*, Ergänzungsband i., 1900. Summary in *Mind*, vol. x.

the association is not correspondingly greater. This result also points to the persistence of a residual charge identical with that to which, as I suggest, the "Perseverations-tendenz" is due.

Other evidence of the residual charge is afforded by the "primary memory-image." This is the name given to the memory-image of an object during a short period immediately succeeding its presentation through any sense-organ. It is found that during the first few seconds the image can be revived by a voluntary effort of attention with peculiar vividness and accuracy, that "after ten seconds a considerable waning has taken place, and after 100 seconds all that is distinctive of the primary image has probably ceased."[1]

Of these various cases that are so readily described in terms of neurin, those which indicate the persistence of a residual charge seem to me the most important, for they show that the activity of the neurones cannot be regarded as a mere propagation and augmentation of an impulse, as is commonly assumed to be the case in peripheral nerve-fibres, or as a wave movement of some unknown medium that sweeps through them, as Richet suggests; but that we must assume that there is generated in the neurones something comparable to a charge of heat or of statical electricity that can be stored up for a time and can leak slowly away.

If, however, the "primary memory-image" and the "Perseverations-tendenz" and allied facts are to be explained as due to a residual charge of neurin, we have to assume that some part at least of the tracts concerned in the production of a sensation and in the reproduction of that sensation in memory are identical. This is a point that has been much disputed. Bain and others have upheld the view of Hume, that the memory-image differs from the sensation only in being less vivid or intense, and they assume therefore that the neural elements concerned are identical. There are, however, few psychologists who would now maintain this view in all its original simplicity. Ward has put together

[1] J. Ward, art., "Psychology," *Encycl. Brit.*, 9th ed.

the evidence that proves that the seats of the sensation and of the memory-image of it are not identical, and he concludes that, "In the complete or apperceived presentation, as in the complete or overt action, we must suppose, then, that the subcortical centres contribute whatever constitutes reality or actuality, of which intensity is one but by no means the sole characteristic."[1] I venture to think that, though this statement comes very near the truth, the view of cerebral activity that I am suggesting will enable us to put the matter in a clearer light, which I will indicate as briefly and as simply as possible. Let the cortical conduction-path concerned in the production of the sensation be schematically represented as simply as possible by a chain of three neurones, *a*, *b* and *c*, afferent, central and efferent respectively. Then, on stimulation of the sense-organ, the excitation spreads through the lower afferent neurones up to *a* and through the chain of cortical neurones, *a*, *b*, *c*, and so out to some motor centre; and the discharges across the synapses *a*—*b* and *b*—*c* constitute the psycho-physical processes that determine the sensation. Now when by association the idea of the same object is reproduced as a memory-image, the neurone *b* is excited not from *a*, but through some neurone of its own level, and it discharges through synapsis *b*—*c* as before. We may then suppose that the psycho-physical processes at synapses *a*—*b* and *b*—*c* are identical in quality and that the peculiar and undefinable quality that distinguishes the sensation from the idea, the presentation from the representation, is due simply to the duplication of the psycho-physical process; or more probably that, while the processes at *a*—*b* and *b*—*c* are essentially similar in quality, they yet differ in some respect, probably in that the discharge of neurin at *a*—*b* takes place at a higher potential than the discharge at *b*—*c*.

In this way we may understand why it is that, while there are so many urgent grounds for regarding the seat of the sensation and of the idea as identical, yet on other grounds such a view is shown to be untenable; there is

[1] "Assimilation and Association," *Mind*, N.S., vols. ii. and iii.

partial identity of "locus," the "locus" of the idea is a part only of the "locus" of the sensation.

This scheme lends itself so well to the description of the neural events underlying mental processes that I am tempted to go on indefinitely, but I must content myself with the exposition of one other important point. We must believe that the presence of a charge of neurin in a neurone, even while its potential is below the threshold of the synapsis, tends in some way to lower the resistance of the synapsis; it may be that the terminal is protruded so as to diminish the the thickness of inter-cellular substance, or, more probably, that some change of state is induced in that substance; suppose, for example, that the charge of the neurone were a charge of heat, and that rise of temperature diminished the resistance of the inter-cellular substance, the effect of an infra-liminal charge would then be parallel to that produced by a charge of neurin. And the facts of sensory attention, especially that aspect of them insisted upon by G. E. Müller,[1] indicate that this effect is probably exerted on both the afferent and the efferent synapses of a neurone; to go back to the scheme of the preceding paragraph, a charge given to *b* from neurones of its own level lowers the resistance of the synapsis *a*—*b* as well as of *b*—*c*; and this effect seems to be in fact an essential part of the facilitation-process. Experimental evidence of the truth of this view is afforded by an experiment carried out by Grossman under Exner's direction, and I must refer to the latter's account of it.[2] The essence of it was, however, to show that, when of three nuclei which normally work together sending out rhythmic series of impulses one is isolated, the other two continue to discharge, but with a slower rhythm and with an increased energy at each discharge; so that, as Exner writes, "We see therefore that the phenomenon of 'Bahnung' may consist not only in charging of centres, but also in the lowering of the discharge threshold."

I will bring this paper to an end by pointing out how the conception of neurin may be applied to the explanation

[1] *Zur Theorie der sinnlichen Aufmerksamkeit.* 1873.
[2] *Op. cit.*, p. 80.

of certain changes in the general state of the nervous system.

The increase of the reflex activity of the cord that ensues when it is separated from the brain, may be explained as follows:—In the normal state a part of the neurin, generated by the sensory neurones leading to the cord under the influence of the gentle stimuli perpetually acting upon them, is led off by the ascending tracts into the brain and slowly filters through it, keeping up its tone without leading to discharges. When the brain is cut off from the cord this wide path is closed and all neurin generated in the afferent neurones passes directly through the reflex conduction-paths of the cord to the muscles, keeping the tone of both cord and muscles at a high level. It is, I think, highly probable that other instances of inhibition of nervous action may be explained along these lines.[1]

On awaking quietly after a night of deep sleep, I lie sluggish and heavy. I seem to be incapable of any muscular effort, in fact I can hardly close my fist, so slight is my voluntary power, and urgent appeals to my moral or intellectual nature may fail to bring me even to a sitting posture. If only gentle stimuli affect my sense organs, I gradually gather energy enough to enable me to crawl to my bath, and then, at the first splash of cold water on my skin, the slackness of muscle and of resolution disappears and I become again a normally active and alert individual. Any violent sensory stimulus, a very loud sound or a very bright light, will produce almost instantaneously a similar effect. During the night neurin has continually leaked away by the efferent nerves from the whole nervous system more rapidly than it is produced by the resting neurones, the potential of the whole nervous system has thereby been reduced to a

[1] Prof. Verworn's paper, "Zur Physiologie der nervösen Hemmungserscheinungen" (*Arch. f. Anat. u. Phys.*, 1900), has come into my hands, through the kindness of the author, since the above was put into type. It is there shown that the inhibition of skeletal muscles is essentially passive inhibition, a cutting off of the stimulating influence proceeding from the motor centres. This fact emboldens me to suggest that in the reciprocal inhibitions of the motor centres of antagonistic muscles we have to do with a leading off of neurin from one path through the opening of a path of less resistance. The reciprocal inhibitions of visual images strongly suggest this view of inhibition.

very low level and evenly distributed throughout the system. A brisk stimulation of sensory nerves then quickly restores my voluntary power and general tone and alertness by causing a rapid production of neurin that diffuses itself throughout the nervous system. Again, if I stay in-doors all day taking no exercise, I grow restless and irritable; the generation of neurin has outpaced the discharge and the general level of potential has become too high. An hour's brisk exercise restores my normal equanimity; the vigorous efferent outflow reduces the general level of potential. Similarly a day spent in steady physical exercise leaves me in that condition of placid enjoyment of sitting still and doing nothing which is best known, perhaps, to mountain-climbers; the outflow has exceeded the generation of neurin, the general level of potential is low.

What exactly is the nature of the process of discharge of neurin across the synapses and what is the part played by the inter-cellular substance can hardly be so much as conjectured, but we must assume that each discharge constitutes, or brings about in the inter-cellular substance, the delicate and specifically differentiated psycho-physical process. And we must further assume that each such discharge leaves the substance so changed that its resistance is permanently lowered. This permanent lowering of the threshold of the synapsis may be conceived to consist in any one of various changes, it may be that the thickness of the layer of inter-cellular substance between the terminals of the nerve-fibrils is diminished, or that its cross-section is increased, it may consist in some state of strain analogous to the strain of a dielectric substance remaining after discharge from a condenser, or in some molecular rearrangement, or more probably in some change of whose nature we cannot at the present time form any conception. This change, which seems to be the physical basis of memory and association, this organisation of the synapses, which corresponds to what Exner calls "Ausfahren" or "Ausschleifung der Bahnen," must be carefully distinguished from facilitation (Bahnung), which is a temporary condition due to the presence of a charge of neurin in the neurones. Exner

himself observes the distinction, but by other writers the two things have been confused.[1]

It may seem to some a grave objection to the ideas propounded above, that they involve the assumption that the psycho-physical process underlying a continuous state of consciousness is a series of processes discontinuous in time. But such a purely *a priori* objection has no validity. It is conceivable that, if a creature whose vital tempo was set at a much higher rate than ours (as is perhaps the case in the gnat and other insects) were capable of being in some way directly affected by our psychical processes, then our psychical processes, thus objectively viewed, would appear to it as a series of pulses of consciousness separated by considerable intervals of time. We have seen that Richet does not scruple to assume that the nerve-wave, which he regards as the physical basis of consciousness or (if we may take his remarks literally) as identical with consciousness, is repeated only about ten times per second; and Herbert Spencer too teaches that our subjectively continuous states of consciousness consist, objectively regarded, in series of "pulses of feeling" corresponding to series of "nerve-pulses."[2]

My proposition, that the synapses are the seats of the psycho-physical processes, involves the corollary that the state of consciousness of an individual at any moment is determined by several, and, in any but the simplest states, by a very large number of psycho-physical processes occurring simultaneously in different parts of the cerebral cortex, the total state of consciousness being the resultant of purely psychical fusion of the several affections of consciousness determined at the several synapses. I am aware that this will not be readily accepted by many minds that have not realised that psychical fusion is a conception absolutely necessary for any but the most superficial consideration of the relation of physical and psychical processes. Psychologists, of course, make use of the terms "psychical com-

[1] I made this mistake in the use of the term "Bahnung" in the essay to which I have several times referred. *Mind*, vol. vii.

[2] "Principles of Psychology," part ii., chap. i.

pounds," "fusion," and "colligation"; but I believe that a large number of psychologists take the same view of fusion as Ebbinghaus, who writes,[1] "Das, was verschmilzt im Falle der Verschmelzung, sind mithin nicht die seelischen Gebilde, sondern die nervösen Prozesse." On the other hand, Helmholtz's theories of sensations of tone and colour involve the assumption of purely psychical fusion, and it is explicitly expressed in the following passage:—"Diese Thatsache ist wichtig, weil aus ihr hervorgeht dass der Inhalt jedes einzelnen Sehfeldes, ohnedurch organische Einrichtungen mit dem des anderen verschmolzen zu sein, zum Bewusstsein gelangt, und dass die Verschmelzung beider Sehfelder in ein gemeinsames Bild, wo sie vorkommt, also ein psychischer Act ist."[2] Exner lays down the principle of the multilocular excitation of a single state of consciousness perfectly clearly and explicitly when he writes:—"Eine Summe von Erregungen in der Gehirnrinde, verschiedene Bahnen betreffende, wirken als ein einheitliches Erregungsvorgang auf das Bewusstsein."[3] Sully[4] and Münsterberg[5] make statements similar to this. In my paper on vision I have used the phrase "psychical fusion" in the sense that is given to it by Helmholtz, and have pointed out that without this assumption it is impossible to explain many visual phenomena in terms of any of the rival theories.[6]

There is, I believe, no greater bar to the progress of our knowledge of mental processes than the frequently displayed reluctance fully to accept and to realise psychical fusion in this sense. This reluctance seems to be due to the deeply-rooted tendency to apply our conceptions of space to psychical processes, and to suppose that the soul can be acted upon only at one place at one time. It is this tendency that has led various authors to assume as the seat of the soul a "punktförmiges Seelenatom," a supreme monad,

[1] Grundzüge der Psychologie," p. 573, Zweiter Halbband, Leipzig, 1902.
[2] *Physiog. Optik.*, S. 921.
[3] *Op. cit.*, S. 236.
[4] "Human Mind," p. 51.
[5] "Grundzüge der Psychologie," p. 556, Leipzig, 1900.
[6] *Mind*, vol. x., section v.

a pineal gland, or a central structureless parenchyma.[1] Of the many considerations that compel us to accept the assumption of a purely psychical fusion I will mention only one. If we do not accept it, we must assume that each state of consciousness is excited by a single complex psycho-physical process. Then, since no one state of consciousness endures more than a moment and no one ever recurs during the life of an individual, we shall have to assume as many million specific varieties of psycho-physical process as the moments of conscious existence experienced by any individual. This is an altogether impossible assumption; so immense a variety of states of consciousness can only result from the varying combinations of the affections of consciousness determined by a relatively few kinds of psycho-physical process. This view presents no difficulty so long as we do not lose sight of the fact that, as Rehmke[2] writes, "Die Seele hat, weil keine Grösse, auch keinen Ort: die Seele ist, aber sie ist nirgends"; the epigrammatic correctness of this statement is, however, a little too rigid, and the truth is perhaps better expressed by Lotze,[3] when he writes: "The soul stands in that direct interaction which has no gradation, not with the whole of the world, nor yet with the whole of the body, but with a limited number of elements; those elements, namely, which are assigned in the order of things as the most direct links of communication in the commerce of the soul with the rest of the world. On the other hand, there is nothing against the supposition that these elements, on account of other objects which they have to serve, are distributed in space; and that there are a number of separate points[4] in the brain which form so many seats of the soul. Each of these would be of equal value with the rest; at each of them the soul would be present, with equal completeness, but not therefore without any distinction; rather we might suppose that at each of them the soul exercises one of those diverse

[1] A suggestion made by Lotze in the *Medizinische Psychologie*, but afterwards rejected by him.

[2] "Die Seelenfrage," *Zeitschr. f. Psychol.*, &c., Bd. ii.

[3] "Metaphysik," English translation, § 295.

[4] *i.e.*, synapses of the cortex.

activities which ought never to have been compressed into the formless idea of merely a single outgoing force." [1]

Another difficulty, to my mind the most serious of all, must be frankly admitted. The synapses of the cord seem to differ from those of the cortex not in kind but in degree only; it might therefore be expected that, if the passage of the excitation-process across the synapses of the cortex constitutes or determines the psycho-physical process, then it should do so in the case of the synapses of the cord also. It may be noted, however, that a similar difficulty meets us even more decidedly if we assign the psycho-physical processes to the nerve-cells of the cortex or of any other part, for, in the case of the nerve-cells, we know of no important difference in structure or function between those of the cortex and those of the cord, whereas, as we have seen, we have strong reason to regard the synapses of these two regions as differing widely in the degree of resistance offered by them to the passage of the excitation-process. The difficulty may be met in either one of two ways. We may suppose that some obscure degree or kind of affection of consciousness is contributed by the synapses of the cord to that complex total resultant of many psycho-physical processes, coexistant in time but separated in space, which constitutes the state of consciousness of an individual at any moment. Or we may suppose that the discharge of neurin across the synapsis constitutes a psycho-physical process only when it takes place at more than a certain potential; as G. E. Müller writes: "But whether the brain-process can possess that peculiarity of being actually a psycho-

[1] In his "Grundzüge der Psychologie" (p. 26), Professor Ebbinghaus asks, "haben wir sachliche Veranlassung, das Seelische in einen unbedingten und scharfen Gegensatz zu bringen zu der Art und Weise des Daseins, die wir als Ausgedehntsein und Materiellsein an Pflanzen, tierischen Leibern, u. s. w., alle kennen?" And he replies, "die Antwort ist nein, wir haben solche Veranlassung nicht." I believe that, on the contrary, the evidence for purely psychical fusion (of which evidence the facts brought together in this essay are an important part), enables us to answer this question with a very decided "Yes," and it seems to me that this evidence furnishes one, and perhaps the only, solid ground for regarding the soul as a being subject to its own peculiar laws, according to which the states or processes excited in it by the brain-processes, interact with one another, and so bring it about that the soul reacts upon the brain-processes to modify them. The same evidence suffices of itself, as it seems to me, to render untenable the doctrine of psycho-physical parallelism.

physical process at any grade of intensity, remains wholly undecided." [1]

This paper may be fitly concluded by the quotation of an authoritative statement by Foster and Sherrington: "The higher psychical processes we may probably regard as being pre-eminently functions of the connexions of the several parts of the cortex with each other." [2] In the preceding pages I have merely attempted to expand and to define more fully the truth conveyed in this statement.

[1] *Pyscho-physik d. Gesichtsempfindungen. Loc. cit.* p. 4.
[2] "Text-book of Physiology," p. 1218.

A CASE OF COMPLETE ABSENCE OF THE VISUAL SYSTEM IN AN ADULT.[1]

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(Read before the College of Physicians of Philadelphia, November 6, 1901).

COMPLETE absence of the visual system is of very rare occurrence, especially in an adult. The study of the case I report in this paper should be of more value than one of agenesia of the visual system in a newborn child, because in the latter portions of the brain not belonging to the visual system are imperfectly developed, and faulty conclusions may therefore be drawn.

The boy, T. S., according to the case-books of the Pennsylvania Training School for Feeble-minded Children, was born in 1878, and was admitted to the School March 12, 1895. At the time of admission he weighed thirty-eight pounds, and his height was 3 ft. 7 in. He was an idiot, absolutely helpless, and very unclean. He could say "mamma," and nothing more. He was passionately fond of music. At the time of admission to the school he appeared to be about 8 or 10 years of age.

I saw him on July 30, 1897, and made a few notes on his condition. I regarded the case at that time as one of cerebral spastic paraplegia of the lower limbs, with absence of the eyeballs. The boy was unable to stand alone, and if he were stood up and not supported he fell backward. When supported he could take a few steps, but in doing so one lower limb was moved in front of the other (cross-legged progression). His knee-jerks were exaggerated, but ankle-clonus was not obtained. He could move his lower limbs while he was sitting, and there was not excessive

[1] From the William Pepper Laboratory of Clinical Medicine (Phebe A. Hearst Foundation).

rigidity of these limbs on passive motion. He uttered only a few monosyllables. His palpebral fissures were very small. He died March 2, 1900, and the necropsy was made two days later. The necropsical notes I shall use only in so far as they are of importance for the understanding of the case.

The body was that of a child apparently about 12 years of age (see fig. 1), though in reality his age was 22 years. No signs of puberty were present. The testicles were not in the scrotum; the right testicle, quite small, was found in the inguinal canal just above the pubes. The genitalia were like those of a child of 10 years. No hairs were found on the pubes, or in the axillæ, or on the face. The circumference of the head was 18¾ inches; the length of the body was 47 inches. The right upper limb could not be fully extended at the elbow, while the left upper limb could be fully extended. Slight contracture of the fingers of each hand was observed, so that the fingers could not be fully extended when the hands were fully extended at the wrists. The fingers could be fully extended when the hands were flexed at the wrists, as the contracture of the flexors of the fingers did not then interfere with the extension of the fingers. The lower limbs were slightly flexed at the knees, and could not be fully extended.

The palpebral fissure of each eye was very small, and the orbits contained only a small amount of what appeared to be fibrous connective tissue. Unfortunately, permission had not been given for the removal of the orbital contents. Nothing resembling an eyeball was seen.

The optic foramina did not exist, and it was impossible to find an opening in the usual situation of these foramina, and here there were merely slight depressions in the skull. No trace of optic nerves, chiasm, or optic tracts could be found (see fig. 2). There was no sign of an external geniculate body on either side, and the thalamus on each side had nothing resembling an optic tract passing from it. The posterior part of each thalamus was rounded and larger perhaps than one would expect to find it in a case of complete agenesia of the visual system (see fig. 3.) The brain was small, firm, and not œdematous. The left ascending frontal convolution in the centre for the upper limb was very small (see fig. 4), to which fact possibly was due the contracture of the right forearm (see fig. 1), because of imperfect development of the nerve fibres innervating this limb. The anterior colliculi of the corpora quadrigemina were fully as large and as well developed as the posterior. The occipital lobes were small, and

the cuneus on each side (see fig. 5) was small, and the calcarine fissure short. The lower olives were covered by thick bands of nerve-fibres—*i.e.*, the external arcuate fibres were unusually well developed.

The spinal cord was small, but not otherwise abnormal.

The brain was hardened in formaline, and the basal ganglia of one hemisphere were cut in serial sections, while those of the other hemisphere were preserved intact as a gross specimen. Sections were made through the cortex of the left calcarine fissure, and stained by thionin and by Weigert's hæmatoxylin method. Frontal sections were made through the right occipital lobe in order that the optic radiations might be studied when cut transversely. The nerves supplying the ocular muscles were also cut and examined.

The cortex of the left calcarine fissure stained by thionin contained many nerve-cell bodies, but they were possibly slightly less numerous than are the cell bodies in sections from the corresponding region of a normal brain, especially in the third, fourth, and fifth layers. The giant-cells of the third layer were represented, but were possibly not as numerous as in a normal brain. The fibres of Vicq d'Azyr of the cortex of the calcarine fissure were not very distinct in sections stained by Weigert's hæmatoxylin method, but they were not entirely absent.

The optic radiations in the frontal sections of the occipital lobe were not entirely absent, but the area occupied by them was not very distinct.

Meynert's commissure was normal, and stained well by Weigert's hæmatoxylin method (see fig. 6).

Careful examination of the serial sections failed to show the slightest trace of an external geniculate body on either side, although the internal geniculate body was well developed. Fig. 6, a photograph from one of the sections, shows the internal geniculate body in its normal size, as well as the inferior posterior portion of the optic thalamus, but the external geniculate body, which should be present in a section at this level, is entirely absent. Comparison with fig. 326 of the *Anatomie des Centres Nerveux*, vol. i., by J. and A. Dejérine, is instructive. This drawing is from a section cut in the same direction as that represented in fig. 6 of my series, and shows the normal size of the external geniculate body and an optic tract in a section from this level. In the section of my series represented in fig. 6 there is no trace of an optic tract.

Some few medullated nerve-fibres are found within the pul-

vinar in my section, but those in the posterior portion of the thalamus are much fewer than those in the more anterior portions. The contrast is very striking, and is represented in a drawing of a horizontal section (see fig. 7), which should be compared with a corresponding section from a normal brain, as shown in fig. 321 of the *Anatomie des Centres Nerveux*, by J. and A. Dejérine.

The habenula in the horizontal sections appears well developed, and the fasciculus retroflexus of Meynert is well stained.

In the photograph the anterior colliculi of the corpora quadrigemina appear smaller than the posterior; but this is merely because the former were further from the camera.

Sections through one-half of the oculomotor nuclei stained with thionin contain many nerve-cell bodies belonging to these nuclei, and these cell bodies stain well with thionin and appear to be normal. They are possibly not as numerous as cell bodies in oculomotor nuclei of a normal brain. Sections through one-half of the oculomotor nuclei were also stained by the Weigert hæmatoxylin method.

Transverse sections of the extracerebral portion of the left oculomotor nerve near the cerebral peduncles stained with ammonium carmine show an apparently normal nerve. The nerve is possibly smaller than an oculomotor nerve from a normal brain, but it contains no sclerotic areas. It contains, however, some areas in which very fine nerve-fibres form distinct groups, but there is no scarcity of large nerve-fibres.

Transverse sections of the right oculomotor nerve are very similar to those of the left oculomotor nerve. The sections stain well by Weigert's hæmatoxylin method.

One of the trochlear nerves in transverse sections, stained either by ammonium carmine or Weigert's hæmatoxylin, appears to be normal, and contains nerve-fibres of good size. The nerve-fibres stain well with the Weigert hæmatoxylin, but in sections so stained the nerve-fibres are perhaps not quite so close together as in the trochlear nerve from a normal brain.

The nerve-fibres of the left abducent nerve stained in transverse sections by acid fuchsin are exceedingly small, much smaller than those of the oculomotor and trochlear nerves, but they stain well with Weigert's hæmatoxylin.

The right abducent nerve in transverse sections stained by acid fuchsin contains many nerve-fibres that are much larger than any of those found in the left abducent nerve, but it contains also many sclerotic areas. Some of the nerve-bundles stained by

Weigert's hæmatoxylin method contain very few medullated nerve-fibres. The contrast between the abducent nerves and the other two ocular nerves on each side is most striking (see fig. 8).

The nucleus of each abducent nerve contains many nerve-cell bodies which, with the thionin stain, appear to be normal.

The nerve-cell bodies of the anterior horns of the spinal cord in the cervical and lumbar regions stained with thionin are normal, but possibly a little smaller than those usually present in the anterior horns. No sclerotic areas are found in the lateral columns, neither are the nerve-fibres in these columns unusually fine in transverse sections.

von Monakow gives as primary optic centres the external geniculate body, the surface of the pulvinar, and the superficial gray matter of the anterior colliculi of the corpora quadrigemina. He says that Stilling, Bernheimer, and Kölliker believed that visual fibres arise also in the subthalamic body, the internal geniculate body, and the tuber cinereum. The anterior colliculi of the corpora quadrigemina in man, according to his view, have a very subordinate *rôle* in vision, and he believes that about 80 per cent. of the optic nerve-fibres terminate in the external geniculate body. My case of congenital absence of the central visual system confirms these views of von Monakow. It shows also that Meynert's commissure and the habenula are not part of the visual system. According to von Monakow (*Gehirnpathologi*) centrifugal fibres—*i.e.*, fibres having their origin in the visual cortex—are present in the optic radiations and pass to the inter-brain and mid-brain. The giant pyramidal cell bodies of the third layer in the cortex of the occipital lobe give off axones, which pass by way of the optic radiations and the arm of the anterior colliculus of the corpora quadrigemina, to terminate in the superficial medullary substance and gray matter about the aqueduct of Sylvius in the anterior colliculus. The Vicq d'Azyr fibres, he says, are often in a double row in the occipital lobe. They come, in greater part, from the fibres of the optic radiations which pass to the third layer of the cortex, but some of the Vicq d'Azyr fibres may belong to an association system. In long-standing lesions of the optic radiations the Vicq d'Azyr

fibres are very atrophic, and the double row of these fibres is scarcely recognisable. In my sections the comparatively few fibres passing to the pulvinar are doubtless the fibres described by von Monakow as arising in the occipital lobe, and in my sections the Vicq d'Azyr fibres are not very distinct.

von Leonowa has examined the microscopical sections from four cases of anophthalmia and three cases of atrophy of the eyeball, but in all these death occurred within a few months after birth. In all her cases the posterior colliculi of the corpora quadrigemina were intact, but the anterior colliculi were affected. von Leonowa[1] concludes from a study of the cases mentioned above that the posterior colliculi of the corpora quadrigemina have no relation to vision, while the anterior have; that the relation of the external geniculate body to the visual fibres is a very intimate one; and that the subthalamic body has no relation to vision. In none of her cases was the absence of the visual system complete, and all the brains were from children in the early periods of extra-uterine life.

In her cases the nuclei of the third, fourth, and sixth cranial nerves were intact, and this integrity is explained by von Leonowa as a consequence of the existence of ocular muscles. The external ocular muscles may be well developed, even though the eyeballs are exceedingly atrophic, as in a case of anophthalmia in a person of 27 years, reported by Haab, in whom the ocular muscles were normal in size and shape, although the ocular bulbs were very small. The nerves to the ocular muscles need not be absent in congenital defect of the visual system. In my case there was apparently no eyeball on either side, but in the small amount of fibrous tissue at the posterior portion of each orbit muscles may have existed, even though they had little or no function. One might suppose that in the case I have reported the third, fourth, and sixth nerves would have been very imperfectly developed in conformity with the law of Gudden, according to which atrophy in a neurone follows injury to this neurone. It should be remembered, however, that in

[1] *Archiv. für Psychiatrie*, 1896, vol. xxvii., p. 53.

my case the nerves supplying the ocular muscles were not injured, and may have been in union with ocular muscles, as in Haab's case.

It has been shown by von Leonowa,[1] in the report of a much-quoted case, that nerve-fibres may develop, although they apparently have no function. She examined a fœtus in which the brain and spinal cord had not developed, and yet the vertebral canal was filled in its entire extent by posterior nerve-roots. Some of the fibres of the posterior roots extended from the lower part of the canal to the upper, as they would have done if they had formed the posterior columns of a normal spinal cord. All the posterior roots ended blindly, and seemed to have sought a spinal cord in which to bury themselves. No anterior roots were found. All the peripheral nerve-fibres present had their origin in the spinal ganglia, and belonged, therefore, to the sensory system. The muscles of the limbs appeared fully normal, although no motor nerves existed. Most of the axis cylinders of the posterior roots were non-medullated or had only a thin medullary sheath. The cell bodies of the spinal ganglia were as numerous and as well developed as in a normal fœtus. The absence of medullary sheaths on the posterior roots, von Leonowa thinks may have been caused by the amniotic fluid, inasmuch as medullary sheaths were present on axones that were covered by the skin.

Gade[2] and Karl and Gustaf Petrén have found posterior roots growing into the spinal canal in complete failure of development of the spinal cord. I am unable to determine from the statements of the latter authors whether in their case or in Gade's the individual fibres of the posterior roots were normal or not.

With the results of these investigations in mind we can understand that nerves might grow equally well into the orbits and persist, even though the ocular muscles were absent. Indeed, von Leonowa and Karl and Gustaf Petrén have shown also that in early life muscles may be developed, although no motor nerves exist, so that the converse may

[1] *Neurologisches Centralblatt*, 1893.
[2] Gade, cited by Karl and Gustaf Petrén. *Virchow's Archiv.*, 1898, B. 151

well be true—*i.e.*, motor nerves probably may exist, although the muscles are not developed. All the ocular nerves in my case, however, were not normal. The two sixth nerves were very imperfectly formed.

Meynert's fibres are asserted by some to be the connection between the optic nerves and the oculomotor centres, although such distinguished cerebral anatomists as Edinger and von Monakow tell us that the anatomical proof for this assertion is wanting. Recent investigations seem to indicate that some of *these* Meynert fibres (for there are more than one band of fibres named after Meynert) belong to a bundle extending into the anterior columns of the spinal cord. In my case the Meynert fibres in the corpora quadrigemina were well developed, although there could have been no reflex fibres from the iris to the oculomotor centres, so that we must conclude that the function of these Meynert fibres cannot be merely to serve the pupillary reflex. I am unable to give the course of the so-called pupillary fibres from a study of my preparations, as the fibres in the region of the corpora quadrigemina form an intricate network.

It is not surprising that cell bodies should be numerous in the cortex of the calcarine fissure in a case of congenital absence of the visual system. Some of these cell bodies have axones which are situated in the optic radiations and terminate in the primary optic centres—*i.e.*, the external geniculate body, the pulvinar of the thalamus, and to a less extent the anterior colliculi of the quadrigeminal bodies, and these fibres probably are not directly concerned in vision. The visual cortex is intimately connected with widely separated portions of the brain, as shown both by physiological and anatomical investigations. The movements of the limbs, the functions of speech, and especially the movements of the eyeballs, are in intimate relation with vision, and important association tracts between the visual area and other parts of the brain can be demonstrated anatomically. The cell bodies of the visual cortex must have axones in relation with remote parts of the cerebral cortex, and, therefore, it is not surprising that these association neurones are developed even in a case of congenital defect of the visual

system. von Monakow represents in his *Gehirnpathologie* (p. 265) a section of a calcarine cortex from a case of lesion of the optic radiations. The giant pyramidal cell bodies of the third layer of the cortex had entirely disappeared, and the third, fourth, and fifth layers were degenerated in a way they were not in my case. It should be remembered that in von Monakow's case the axones destroyed were those of cell bodies situated in the calcarine cortex, and that atrophy of these cell bodies probably followed the injury of their axones. This did not occur in my case, and probably the nerve-fibres present in my specimens in the optic radiations arose in the occipital lobe. It is difficult to understand what the function of these fibres can be.

Although the individual described in this paper was an idiot, we should be cautious in attributing his deficient mental development to the absence of his visual system. It would seem probable, however, that the entire brain was in a state of imperfect development.

The spastic paraplegia of the lower limbs deserves mention. The nerve-cell bodies of the anterior horns in the lumbar cord were normal by the thionin stain, except that they were not so large as the cell bodies of this region usually are, and the lateral columns did not contain sclerotic areas. This is not the first time I have found an apparently normal spinal cord in congenital spastic paraplegia, and I think the explanation is to be found in an imperfect development of the motor neurones. A deficiency from birth in the number of these neurones would probably have the same result as partial destruction of the central motor tracts, and in the absence of the development of motor neurones there would be no sclerosis, inasmuch as no space existed to be filled by proliferation of the neuroglia. Indeed, sclerosis may be entirely absent if destruction of nerve fibres occurs in early life, because the growing tissue of the spinal cord fills the space left by degenerated fibres, and proliferation of the neuroglia does not occur. I have seen the spinal cord in a case of infantile hemiplegia presenting little or no sclerosis, although the person from whom this cord was removed had lived many years after the

paralysis had developed. The case to which I refer was in Dejérine's service. In two cases of congenital spastic paraplegia of the lower limbs I believed I was able to detect an unusual fineness of the nerve-fibres of the pyramidal tracts, but I was unable to do so in this case, T. S.

I am indebted to Dr. de Schweinitz for the reference to a case reported by W. S. Little[1] in 1885, in which congenital absence of some portions of the visual system was found. The brain was from a child of ten years of age, who had been an inmate of the Pennsylvania Training School for Feeble-minded Children, and the specimen was examined by A. W. Wilmarth. This specimen and mine have therefore come from the same institution. The child had had bi-temporal hemianopsia. There was no optic commissure, but a small projection was found on the inner side of each nerve where the commissure should have been. These projections did not touch by at least one-quarter of an inch. A complete study of the specimen does not seem to have been made.

A still more important case, and one more closely resembling mine, has been reported by Haab.[2] His specimen was from a girl, twenty-seven years old, who had the development of a child of fourteen or fifteen years, and from her appearance had not reached puberty. She was an idiot, and had much difficulty in walking. The optic chiasm was entirely absent, but the optic tracts were present as small bands. The oculomotorius, abducens, and trochlearis appeared to be normal, and yet the merest trace of an eyeball was found. The muscles of the orbits appeared to be normal. The external geniculate body was entirely absent, but the internal geniculate body was of normal size. The corpora quadrigemina were of normal size macroscopically. The optic foramen was almost obliterated. The pulvinar of the optic thalamus was smaller than in a normal brain.

The conclusions that may be drawn from my case are:

[1] *Transactions of the American Ophthalmological Society*, 1885, p. 367.
[2] *Beiträge zur Ophtalmologie als Festgabe*. Friedrich Horner, 1881, p. 131.

(1) The chief "primary" optic centre is the external geniculate body.

(2) The pulvinar of the optic thalamus is also an important "primary" optic centre.

(3) The anterior colliculus of the quadrigeminal body in man has an unimportant relation to vision.

(4) The subthalamic body, the habenula, the internal geniculate body, probably are not part of the visual system.

(5) The cortex of the calcarine fissure may contain nearly the normal number of cell bodies, even though the visual system may be undeveloped.

(6) The nerves to the ocular muscles and their nuclei may be developed, even though the visual system is absent.

(7) Congenital spastic paraplegia may be the result of deficient formation as regards number or size of the neurones of the central motor system, even though such a deficiency may be difficult to detect by the microscope.

FIG. 1.

A photograph of T. S., taken after death. The right upper limb, the fingers of both hands, and the lower limbs are slightly contractured. The palpebral fissures are small.

FIG. 2.

The occipital lobes have been cut away. A portion of the right temporal lobe has also been cut away in order to show the complete absence of the visual system. The internal geniculate body (A) is well shown, but no trace of the external geniculate body or of an optic tract or chiasm can be found.

FIG. 3.

The optic thalami are well shown. The anterior colliculi of the corpora quadrigemina, especially the right colliculus, appear smaller than the posterior, because the former were further from the camera.

FIG. 4.

The left ascending frontal convolution in the centre for the upper limb (B) is very narrow.

FIG. 5.

The upper part of the right cerebral hemisphere, showing the small cuneus (C).

FIG. 6.

Photograph of a horizontal section of one cerebral hemisphere, showing Meynert's commissure (B) well developed, and also the internal geniculate body (C), while the external geniculate body is entirely absent.

FIG. 7.

A drawing of a horizontal section through the basal ganglia of one hemisphere. The fibres entering the posterior part of the optic thalamus are few in number.

FIG. 8.

Drawings of transverse sections through portions of the abducent nerves. The upper drawing represents the fine fibres of the left abducent nerve, and the lower drawing the sclerotic areas in the right abducent nerve.

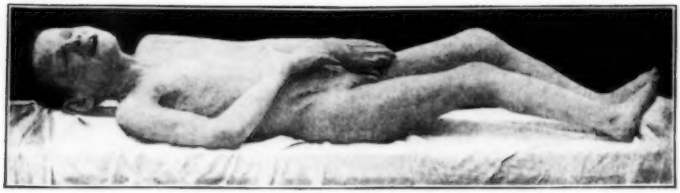

FIG. 1.

FIG. 2.

FIG. 3.

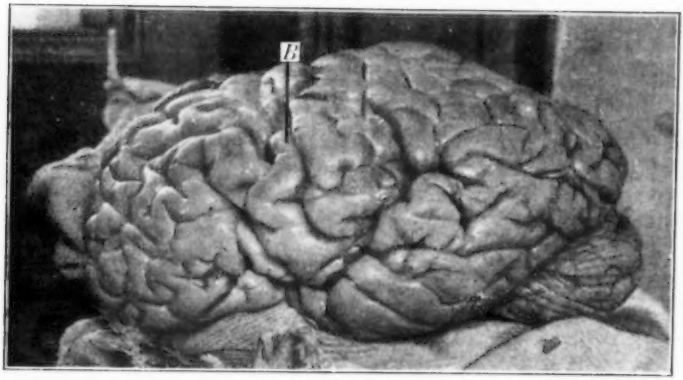

FIG. 4.

FIG. 5.

Fig. 6.

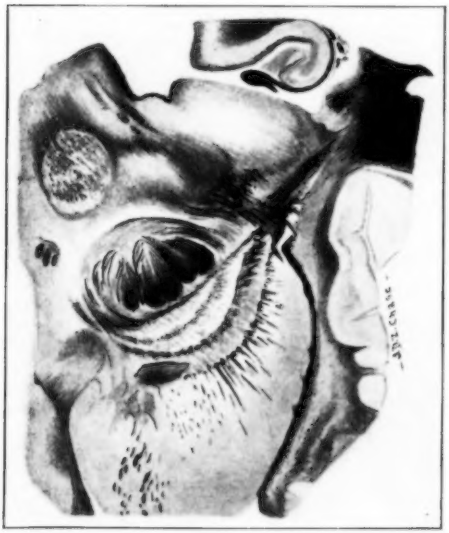

Fig. 7.

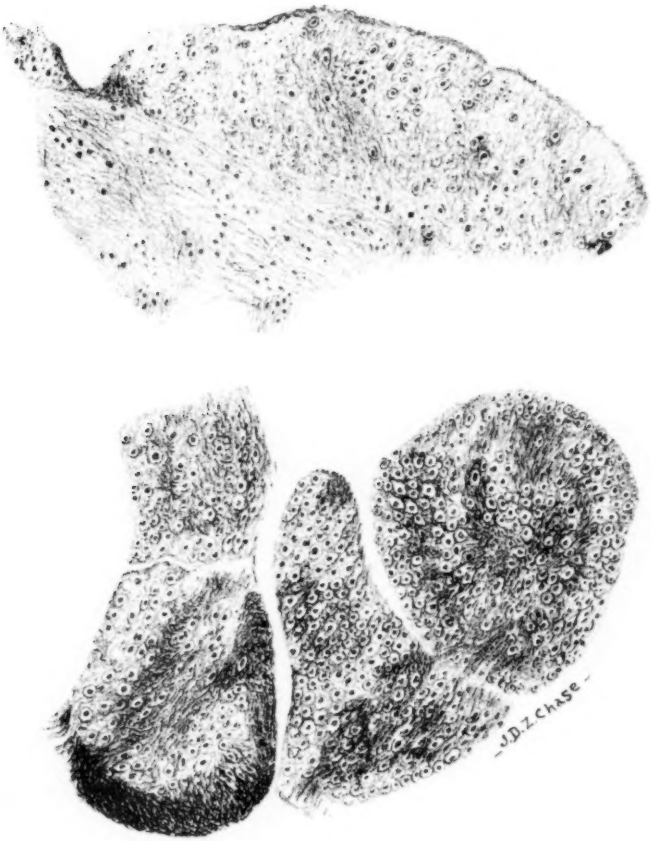

Fig. 8.

THE AREAS OF CUTANEOUS DISTRIBUTION OF THE 1ST SACRAL AND 5TH LUMBAR NERVES AS ILLUSTRATED BY TWO CASES OF HERPES ZOSTER.[1]

BY T. ALDOUS CLINCH, M.D.
Nottingham.

CASE 1.—*In the Area of the First Sacral Nerve.*

THE extreme rarity of eruptions of herpes zoster in this area invests the following case with more than common interest. Head has described but one in all his great experience, and that associated with simultaneous affection of the fifth lumbar area.

I am not acquainted with any other case, but my opportunities of studying the literature have been very limited.

The patient I attended for Dr. Collins, Nottingham, to whose generosity I am indebted for permission to publish the following account.

History.—Patient was a domestic servant of rather low type, about 50 years of age, and probably syphilitic. On August 29 the right great toe became numb; on the 30th it was red and painful, and on the 31st black. At this date the eruption appeared: a small circular patch about the size of a florin, slightly external to the posterior superior spine of the ilium; a second just posterior to and immediately below the great trochanter, about one and a half long by one inch broad; a third below and slightly posterior to the head of the fibula. All these patches were of moderate severity, and not preceded by the sensations and signs shown in the foot.

Present state.—When I first saw her, five days after the onset of the attack, an area of the foot—comprising the outer

[1] This paper was read before the Nottingham Medico-Chirurgical Society, December 4, 1901.

half of the sole, the tread of the foot, and the terminal phalanx of the great toe on both plantar and dorsal aspects—was reddish black; it ached and was painful and hot to the touch. Through the thickened epithelium appeared a few dimly seen yellowish spots. I was shown no more at that visit, and my hasty diagnosis was a diffuse superficial cellulitis. I am sure a correct

CASE 1.

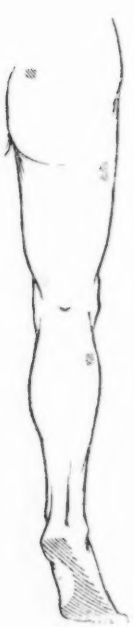

FIG. 1.
Area of distribution of herpes in Case 1.

FIG. 2.
Area of distribution of small sciatic (copy from Quain).

FIG. 3.
Probable area of distribution of first sacral on thigh.

diagnosis was possible, and I hope I could make it another time.

Treatment ordered was poultices with recourse to the "knife" later, if necessary.

Progress.—The next morning the other areas were displayed to me and a mistaken diagnosis was no longer possible. The

poultices had afforded much relief to the painful symptoms in the foot.

Constitutional malaise, which was very severe for the first few days, had also somewhat subsided. Future progress presented no conditions worthy of further attention.

I very much regret not having made a complete examination of the nervous system, but its importance did not occur to me till too late.

Remarks.—The further and more exact anatomical diagnosis of the primary lesion appeared at first sight difficult, but I hope that my reasons for locating it as I have indicated in the title will carry conviction to others besides myself.

The first point that struck me was that the foot lesion corresponded almost exactly to the area allotted by Head to the first sacral; but above and beyond this area were three areas which did not coincide with any one known area. Dr. Head has laid it down that there are three maximum points of attack in a case of herpes: (1) that corresponding to the posterior primary division; (2) that corresponding to the lateral branch, and (3) that corresponding to the anterior branch of the anterior primary division of the nerve.

The sacral patch corresponds fairly closely with the area supplied by the posterior primary division of the first sacral, as displayed by the method of dissection, and it is, in my opinion, not assuming too much to consider that this patch is due to lesion of the first sacral. It moreover lies immediately above an area allotted by Head to the third sacral.

In normal cases the small sciatic nerve-trunk draws its supply of fibres from the second, third, and first sacral nerves, the order given being that of the predominence of the fibres. Now the area of skin supplied by the small sciatic, as shown in the last edition of Quain, corresponds very closely with that given by Head as the combined area supplied by the second and third sacral. Head, however, gives no area in this locality for the first sacral, which obviously must have some skin to supply, seeing that some fibres from it enter the small sciatic trunk. The second sacral being arranged pre-axially to the third sacral, it is not illogical to assume that this first sacral area, *which must*

exist, will be on the pre-axial border of the second sacral area. Now it is on this border that we find the two remaining patches of herpes in this case, and I believe that they indicate the existence of a narrow strip of skin along the pre-axial border of the second sacral area, which is supplied by the first sacral, that they may indicate its extent upwards and downwards, and that it is probable that this area is very narrow in its mid portion, and may be actually separated in two.

If my hypotheses are correct the first sacral supplies an area of skin corresponding to its posterior primary division, close to the posterior superior iliac spine, a narrow strip of skin extending from the great trochanter to below the head of the fibula, corresponding to the lateral branch of the anterior primary division, in addition to the area on the foot and leg already described and figured by Head.

For my sketches I am indebted to Dr. Ferraby, to whom and Dr. Collins I have to tender my warmest thanks.

CASE 2.—*Occurring in the Area of the Fifth Lumbar.*

I am indebted to the kindness of Dr. Ferraby, of the Branch Dispensary, Nottingham, for this case. Dr. Ferraby, in addition to allowing me to report the case, has been so good as to draw a most graphic sketch of the parts affected.

It is of special interest on one point, and is also sufficiently rare to be worthy of record. Head has reported two uncomplicated cases and one complicated with simultaneous affection of the first sacral.

The details briefly are as follows:—

The patient, an old woman of 70, on Thursday, October 17, experienced the premonitory symptoms of the disease over an area on the buttock, some inches from the cleft, and on a level with its commencement. A week later the rash appeared: two small areas of vesicles separated by an interval of about one inch, and simultaneously pains commenced in the leg area, which corresponds so closely with that given by Head that it needs little further description. The rash—which, according to her account, came out a week after the buttock area—perhaps hardly

descended so low on the foot, while it ascended to the level of the outer condyle of the femur, a somewhat higher level than that given in Head's diagrams, belonging, therefore, to class called by him "prefixed."

I obtained the foregoing history a week later still, when I saw her for the only time, and on that occasion she still had some

CASE 2.

pain and neuralgia, but the vesicles had dried and the scabs had been removed for the most part. There was no change in sensation to touch, pain, heat and cold, as compared with the other limb. The methods used were somewhat coarse. The knee-jerk was markedly impaired, that of the other limbs being, I believe, increased to some slight degree.

This cannot be attributed to interference with fibres connected with the path of the reflex arc; for though the knee-jerk is not a true reflex, yet the reflex arc must be complete in order that it may be present.

The interference with the fifth root must be supposed to have lowered the tonus on the same side of the spinal cord, while it may have possibly slightly irritated the opposite side, thus illustrating the law that stimulation, irritation and depression are effects resulting from the same cause acting with different degrees of intensity. I could detect no abnormality in the plantar reflex.

The point perhaps of most interest is the buttock area; as in the first case it has not been previously described. I regard it as indicating the area supplied by the posterior primary division, and it further helps one to see that on the buttock, which up to now has seemed such a medley of areas, there is the same order and systematic arrangement as in the other parts. Dr. Head tells us that many of these lower areas have been hardly more than guessed at.

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TWO CASES OF ASTEREOGNOSIS.

BY THEODORE DILLER, M.D.

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As a contribution to the study of the perversion of the stereognostic sense I desire to place on record the two following cases :—

Case 1.—A single man, aged 40, was nineteen years ago struck in the left parietal region by a brick, which badly fractured the skull, necessitating the removal of a considerable piece of bone. He immediately lost consciousness, and he has no memory for the four months succeeding the accident.

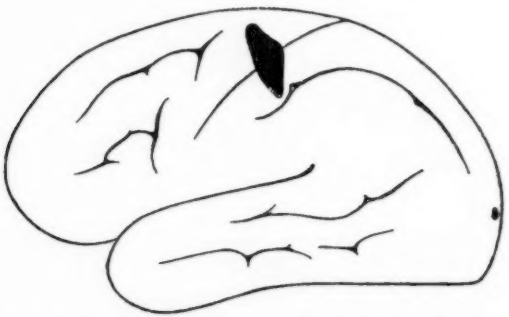
He sustained a left hemiplegia, which disappeared five months after the accident (one month after he "came to"). But a certain "clumsiness" of the right hand remained and has persisted up to the present time, so that while the hand became as strong as before the accident, its usefulness was, in a large degree, lost. A few months after the accident he was seized with an attack of typhoid fever, from which he made an excellent recovery. One year after the accident he contracted a chancre, which was followed by secondary symptoms.

For six or seven years past he has had difficulty in passing his urine, being compelled to make extra muscular efforts to accomplish the act and only partially succeeding in emptying the bladder. The stream is small and often interrupted. For the past two or three years his sexual powers have been declining, and during this period his vision has been steadily failing. Two years ago his left eye "turned out" rather suddenly, but after several weeks it returned to the normal position. For eighteen months past he has staggered in walking, which circumstance gave rise to the suspicion that he was drunk.

Examinations were made on September 6 and several subsequent dates. In the left parietal region there is a deep depression in the skull about 3 inches long, tapering at both ends, and about 1 inch wide in the middle just over the middle of the

central convolutions, extending from 1 inch to the left of the median line, downward and slightly backward, crossing the line of the Rolandic fissure about its middle like the letter X (see figures).

The knee-jerks are absent. There is a moderate degree of static and locomotor ataxia. Eyes:—Pupils: right, 3½ mm.,



A rough diagram intended to illustrate the relationship of the depression in the skull to the central convolutions in Case 1.

Photograph of patient in Case 1, with a piece of white paper placed over the skull depression, to indicate roughly its area and position.

left, 3 mm. in diameter. Neither react to light, but both react slightly to accommodation. Vision is very dim. In both eyes advanced optic atrophy was discovered (Dr. Curry). When examined, stripped, no loss of motion or atrophy can be discovered. He recognises promptly in all parts of his body,

contact, pain, heat and cold. He is not able to recognise objects placed in his right hand, *e.g.*, coins, corks, watch, bottle, pocket-book, key, ring, card, pocket-knife and handkerchief. When these objects are placed in the left hand he recognises them instantly. On the palm of the right hand he recognises the two points of the æsthesiometer as such only when these are at least 60 mm. apart on the fingers and 80 mm. apart on the palm, and at still greater distance apart on the arm and back of the hand and on forearm. But with the right hand he tells many of the qualities of objects placed within it, *e.g.*, hard, soft, smooth, rough, wet, dry, heavy, light, warm or cold. Indeed, he estimates small differences in weight and temperature correctly. Even with the hand lying prone upon a table he estimates accurately small differences in weight (*e.g.*, dime and quarter). With either foot he recognises the length or point of a pencil.

When the fingers and wrist of the right hand are passively placed in different positions of flexion, extension, ab- and adduction, he is unable to reproduce these positions in the other hand. But he promptly reproduces such positions in the right hand when made in a similar manner in the left. Positions of the shoulders and elbows are reproduced correctly on either side.

REMARKS.—That the case is one of tabes, that the astereognosis was produced by the head injury, and that the astereognosis is not a symptom of the tabes, all seem clear. The extensive skull depression seems to be filled in with fibrous tissue. The head injury must have been very severe indeed and the general commotion extensive. That the central convolutions, in their middle third, were the brain structures which were most severely involved by the traumatism is borne witness to by two facts: (1) the history of hemiplegia following the injury, and (2) the location of the skull depression. That the pathological processes induced by the injury were largely dissolved is borne witness to by the fact that after hemiplegia had existed five months it then disappeared. But that certain pathological changes of a permanent character remain seems clear when the long persistence of the astereognosis (over eighteen and a half years) after the disappearance of the hemiplegia is considered. Where are these changes which are producing the astereognosis in this patient? While this question cannot,

of course, be answered positively, yet the probabilities point, with very considerable force, to the middle region of the central convolutions as the chief seat of these changes. But how widespread they may be can only be conjectured.

In brief, the evidence in this case, so far as it goes, points to the middle third of the central convolutions as the cortical location of the astereognosis sense or judgment, and that cells other than the motor cells of this region are concerned in the appreciation of this sense. The case also affords evidence in favour of the view that all sensory impressions are received in cortical regions other than the central convolutions (pain, contact, temperature senses intact).

If the theory of Walton and Paul[1] be adopted as a working hypothesis, this case may be explained upon it. According to this hypothesis the "lower grade" of sensory memories (contact, pain, temperature) for the hand and arm are received in the cortical area posterior to the Rolandic convolutions, *i.e.*, in the parietal or post-Rolandic region, while in the middle portion of the central convolutions the various sensory impressions coming from the arm to the post-Rolandic area are assembled to form the stereognostic sense or judgment.

Dejérine[2] says that it never occurs that all forms of sensation, superficial and deep, are perceived when astereognosis is present, *i.e.*, that astereognosis alone is never seen. Certainly in both the cases here presented the reception of one sort of so-called sensory impulse was strikingly defective, viz., that of the spacing sense, while another, the position sense, was somewhat altered, although not absent. Dercum[3] found spacing sense almost uniformly absent in a large number of cases of astereognosis which he studied, and concludes that loss or impairment of the spacing sense is the most important factor in determining astereognosis. He also concludes that "next in importance to the spacing sense appears to be the knowledge of the position of the fingers and ataxia of movement." Commonly "spacing

[1] BRAIN, Autumn, 1901, pp. 432-433.
[2] *Semiologie du Système Nerveux*, p. 890.
[3] *Jr. Nerv. and Ment. Diseases*, 1900, p. 569, *et seq.*

sense" and "position sense" are absent (or defective) when astereognosis is present; while the ability to perceive the "lower grade" of sensory impressions (pain, contact, temperature) is present. This is true of both my cases. But neither the "spacing" or "position" senses, it seems to me, are simple sensory impressions, as, *e.g.*, pain, temperature, contact, *but rather expressions of stereognosis*, and that their loss is due to the inability to properly assemble and coordinate sensory impressions received, and not factors in producing it. *In other words, "spacing" and "position" senses are component elements of the stereognosis judgment.* If this be allowed it could safely be said that all forms of primary sensory perceptions are present in the case related, unless, perhaps, muscular sense, and we cannot be sure this is absent.

Case 2.—A metal polisher, aged 35, has been married eleven years. Fifteen years ago he contracted syphilis. For many years he has used tobacco and alcohol to excess.

In June, 1900, he was, without warning, seized with a general convulsion of three hours' duration accompanied by loss of consciousness. A month later a second general convulsion occurred, which lasted five hours. A sensation of dizziness and "darkness before the eyes" gave him five minutes' warning of the approach of this attack. In October following he had a third similar convulsion of fifteen minutes' duration, ushered in by the same aura. Last November he was struck, while in a brawl, a severe blow on the head. Ever since this time he has had "numbness" in the right face, arm and leg. In January he began to suffer from severe and persistent headaches, which continued up to August, since which time he has been free from them. This headache, at times, was so severe that he was compelled to go to bed. Vomiting was not a symptom.

Examination on April 26 discovered optic neuritis in the right eye (Dr. Curry). The left nerve could not be examined because of a corneal opacity resulting from an old injury. Stripped, he exhibited no muscular atrophy. Dynamometer: right, 83; left, 76. The knee-jerks were both present, the right more prompt than the left, but scarcely exaggerated. The Babinski toe reflex was absent. Station and gait seemed normal.

Sensation.—From all parts of his body sensations of contact, pain and temperature are normally perceived. As a rule, he is

unable to recognise various objects placed in his right hand. He was tested with various coins, paper money, pencil, bottle, ring, watch, cards, cork, pocket-knife, key, &c. He could tell very much concerning objects without being able to say what they were. He appreciated even slight degrees of heat and cold and small differences in weight. He could tell whether objects were large or small, smooth or rough, hard or soft, wet or dry. He correctly recognised a pocket handkerchief and knew that a card was pliable, and suspected it might be a piece of leather. All these objects, when transferred to the left hand, were quickly recognised.

When the right hand and fingers were placed in various positions of flexion, extension, ab- and adduction, these positions could usually be imitated by the other hand, but not always and never without hesitation and apparent conscious effort. This was in strong contrast to the prompt and invariably correct reproductions in the right hand and fingers, when the left were in a similar fashion passively moved. The ability to recognise two points as such ("spacing sense") showed that they could be so recognised only when 40 to 60 mm. apart on the palmar surface of the fingers; and when 60 to 80 mm. apart on the palm itself. On the right arm and forearm two points could be recognised as such when 30 mm. apart in the transverse direction, but only when 80 to 125 mm. apart in the longitudinal direction.

On October 29 last the optic neuritis had subsided, leaving an atrophy behind (Dr. Curry). The urine was found to be normal.

Remarks.—A consideration of the convulsions, severe headaches, optic neuritis going on to atrophy, taken together, clearly point to a gross intracranial lesion; and the probabilities, I believe, favour a tumour rather than a diffuse syphilitic process. The convulsions cannot be considered as giving any hint as to the location of the lesion, since they were always general in character. Can the astereognosis be considered as a localising symptom? It may at least, I believe, be so regarded in that it indicates that the disease process is situated in the left cerebral hemisphere. Does it indicate anything more? I believe that when the absence of cranial nerve involvement in the case is considered in connection with the accumulated evidence favouring the central or parietal convolutions (or both) as the seat of the

stereognostic sense it would be reasonably safe to diagnose a lesion of the cortex, involving either the Rolandic or parietal region or both these areas of the cortex; and with the evidence afforded by my first case and from a consideration of the arguments adduced by Walton and Paul[1] I should favour trephining over the region of the middle third of the Rolandic region, in case of operation, rather than over the post-parietal convolutions, in which region it is held by Mills[2] impressions of cutaneous sensation and muscular sense are perceived, and where stereognostic judgment takes place. Certainly the first case here reported argues strongly against Mills' view that the cortical centres for stereognosis are entirely separate from the motor senses proper (see diagram).

An operation may yet be performed on this patient, a study of whose case affords an example of the extreme desirability of the ability to localise the stereognosis sense, considered from the purely practical point of view alone.

[1] *Loc. cit.*

[2] "Text-book on Nerv. and Ment. Dis.," p. 341 and p. 257 *et seq.*; also *Journal Nerv. and Ment. Dis.*, November, 1901, p. 599 *et seq.*

Reviews.

Vorlesungen über die pathologische Anatomie des Rückenmarks. Unter Mitwirkung von Dr. SIEGFRIED SACKI, Nervenartzt in München, herausgegeben von Dr. HANS SCHMAUS, A. O. Professor und 1. Assistent am patholog. Institut in München. (J. F. Bergmann, Wiesbaden, 1901.)

THIS book, which has already attained a wide popularity in Germany, is based upon a course of lectures delivered by Professor Schmaus upon the pathological anatomy of the spinal cord. With the collaboration of Dr. Sacki the original material of those lectures has been amplified and extended so as to present a very complete account of the present-day views on the subject.

The various diseases of the cord are approached mainly from the anatomical standpoint, clinical considerations being but briefly referred to. Yet the clinician who studies this work cannot fail to gain much useful information.

Of the twenty-one lectures which constitute the book, the first four are devoted to the general anatomy of the cord and its various elements. The chief spinal tracts are enumerated, and the origin, extent and termination of each is discussed. With regard to the neurone theory, although the authors do not consider its position altogether unassailable in the light of recent researches, yet it affords a useful basis on which to study system degenerations.

The structure of the various types of nerve-cells is clearly described and illustrated, together with the changes which they undergo under the influence of fatigue, fevers, organic and inorganic poisons, &c. The term "chromatin," as applied to the Nissl granules of the nerve-cells, is deprecated, and Lenhossek's term of "tigroid substance" is adopted instead—the former name belonging strictly to the nuclear chromatin, an entirely different substance. Tigrolysis is a condition the result of reaction of the nerve-cells to various influences, not a primarily degenerative process. It may or may not be accompanied by disturbance of function.

Wallerian or secondary degeneration of nerve-fibres, the result of interruption between the axis-cylinder process and the nerve-cell or of disease of the cell, is described and contrasted with primary degeneration of nerve-fibres, which occurs in the absence of both of those conditions. Many, if not all, of the primary degenerations are regarded as due to the influence of toxins, and occur in various systems of fibres, in single tracts, or in groups of tracts habitually co-ordinated in function. Examples of such primary toxic system diseases are afforded by ergotism, lathyrism, chronic alcoholism, lead and other metallic poisonings, blood diseases, auto-intoxications, and cachectic diseases.

Three lectures are devoted to the pathological anatomy of tabes dorsalis. After a description of the spinal degenerations, essentially of exogenous tracts, in this disease, the elective distribution of the various degenerations is discussed. The posterior root-fibres do not degenerate equally or simultaneously; some are affected earlier than others. Some observers account for this by the various functions of these fibres, whilst others associate it with the different times during fœtal life at which the various fibres of the posterior roots become medullated. The extra-medullary changes in tabes dorsalis in the peripheral nerves, the trophic changes in bones, joints and skin, are described, but the association between tabes and general paralysis of the insane is somewhat briefly dismissed. The meningitic theory of tabes, which refers the condition to compression of the posterior roots by a syphilitic affection of the membranes, is discussed, and it is pointed out that such meningitis, though frequent, is by no means constant, even in advanced cases; moreover, the symmetry of tabetic degeneration is in marked contrast to the degenerations which commonly follow meningeal affections.

The pathological appearances in muscular dystrophies, in spinal muscular atrophy, in chronic anterior poliomyelitis, in amyotrophic lateral sclerosis and in primary lateral sclerosis are then described. A distinction is drawn between progressive muscular atrophy and chronic anterior poliomyelitis, the latter being characterised by the fact that paralysis precedes muscular atrophy and is not contemporaneous with it as in progressive muscular atrophy, and also by the course of a case of chronic anterior poliomyelitis being one of definite waves rather than a gradually progressive one, and finally by the remissions which occasionally occur during its course.

After a chapter on anæmic degenerations, emboli, hæmor-

rhages and œdema, three lectures are allotted to the difficult subject of acute myelitis, a clinical term applied to all cord affections not confined to any particular systems or constituents of the cord, but affecting sensory, motor, trophic and reflex functions in a wholesale, irregular, non-systematic fashion. The term "myelitis" includes not only inflammatory processes, but also certain non-inflammatory ones. It is not applied to the results of traumatism, to anæmic necrosis, or to hæmorrhages or emboli. Three chief pathological types of myelitis are described, viz. :— parenchymatous degeneration, small-cell infiltration and softening. Parenchymatous myelitis is characterised by swelling and œdema of the connective-tissue elements, with degenerative changes in the nervous elements ; small-cell infiltration shows a preponderance of vascular changes, of hyperæmia and of leucocytic infiltration, with occasional hæmorrhages, whilst softening consists in the reduction of a portion of the cord to a pappy or fluid consistence, the broken-down area being made up of proliferated leucocytes, together with detritus of nerve elements. This last variety is often associated with vascular thrombosis. If capillary hæmorrhages occur into such an area we have the so-called red softening. Unfortunately corresponding clinical distinctions between these different pathological forms cannot at present be drawn.

Poliomyelitis is regarded as an affection of infective origin, distributed in the area of the anterior spinal artery. Landry's paralysis is also due to an infection, such a view being supported by the concomitant enlargement which occurs in the spleen and mesenteric glands during the disease. The varying pathological appearances which have been found, tigrolysis of the anterior cornual cells, polyneuritis, &c., and the occasionally entirely negative results observed, are to be explained by the fact that the poison which produces the symptoms does not always have time to produce recognisable microscopic changes before a fatal result ensues.

After an account of acute disseminated myelitis and purulent myelitis, a description of chronic myelitis is given, and the two chief types are shown to be, one in which there is a condition of primary degeneration with consequent sclerosis, and another in which a primary softening is followed by cicatricial contraction.

The morbid anatomy of insular sclerosis is excellently described and illustrated, and the various theories regarding its pathogenesis are referred to. The writers come to the conclusion that cases of insular sclerosis are to be classified into two groups, the one a secondary degeneration following an acute disseminated

myelitis of inflammatory, syphilitic or vascular origin, the other a primary form of degeneration, the result of incomplete or anomalous development, or of hypoplasia of the nerve elements.

The various combined system degenerations and the traumatic affections of the cord are discussed. Under the subject of regeneration after injuries it is pointed out that only a few new fibres are formed in the region of the lesion, and those are in close association with neurilemma cells, thus belonging to the type of peripheral rather than to that of central nerve-fibres. Spinal contusion and spinal shock are described, and chapters on compression-myelitis of tuberculous and other origin, on syphilitic and tuberculous disease of the cord, on developmental anomalies, syringomyelia and spinal tumours, conclude a volume of remarkable interest.

The book is copiously illustrated by clear and well-chosen drawings, many of them coloured, the execution of which leaves nothing to be desired. As a *résumé* of our present knowledge of the pathology of the spinal cord Drs. Schmaus and Sacki's work merits the highest praise.

PURVES STEWART.

Studii Clinici ed Anatomo-Patologici sull' Idiozia (Clinical and Anatomo-Pathological Studies on Idiocy). By Dr. PELLIZZI, Teacher of Psychiatry at the University of Turin. (Turin, 1901, pp. 275.)

THIS book is divided into three parts:—The first and most important part treats of idiocy with tuberose sclerosis; the second gives a classification of idiocy; and the third describes shortly epilepsy and idiocy.

Dealing with the first part, we find that idiocy with tuberose sclerosis occupies 200 out of 275 pages of which the book consists. After a short introduction, the author gives briefly an historical account of idiocy with tuberose sclerosis, in which the labours of Bourneville and others who have written on the subject are fully recognised. A description is given of twenty-two cases which the author had collected, five of them being described by Bourneville and his assistants. He then proceeds to give an account of three cases which had come under his own observation—the family history, personal examination, course of the disease, and autopsy.

with the microscopical appearances seen by the Golgi and Weigert-Vassale methods, being given in each case. The second and third cases are more fully described, and the distribution of the tuberosity, the state of the neuroglia, nerve-cells, white substance, and blood-vessels of the brain and of the spinal cord are given at length. The author presents a table of the twenty-five cases of tuberose sclerosis: the name of the author comes first, then the sex and age of the patient, facts concerning hereditary predisposition and other etiological conditions, physical anomalies, the commencement of the case, the motor and psychical symptoms and the result follow, and the principal anatomo-pathological facts are summed up at the end of the table. On looking through it we find there is a history of hereditary predisposition in every case, but other etiological conditions, such as emotion, fright, and epileptic attacks of the mother during pregnancy, were only found in eleven cases. A curious fact is noticeable, viz., that the chief physical anomalies were renal tumours, which were present in sixteen of the twenty-five cases. The motor symptoms consisted entirely of epileptic attacks. With regard to etiology, the external physical and psychical causes and internal causes are mentioned, and idiocy with tuberose sclerosis is compared with other forms of the affection. The symptoms of the disease can be described in a few words. They consist of psychical and motor alterations; under the former head is arrest of mental development, which varies from semi-imbecility to the most profound idiocy; under the latter, ordinary convulsions, rarely partial epilepsy, and sometimes simply choreic movements, but no paralysis nor paresis, and no contraction of limbs. The clinical differential diagnosis between the affection under notice and sclerosis in infants, atrophic sclerosis, and epilepsy is given, and the macroscopical and microscopical appearances of the sclerosis are fully described. To the naked eye tuberose sclerosis consists of islands of sclerosis, numbering sometimes as many as thirty or forty, scattered throughout the hemisphere. They (the islands) are white or slightly grayish and shining in the peripheral parts, but towards the centre the gray is more intense in colour, and has more of a gray-yellowish tint. In consistence they are elastic at the periphery, becoming more resistant towards the centre, finally assuming a cartilaginous hardness. As regards the microscopical appearances, it should be noted that each tuberosity or hypertrophied sclerotic plate consists of nerve elements and neuroglia. In the central or hardest part, the nerve-cells are few in number

and sometimes almost completely absent, but there is a dense intertwinement of fibres of the neuroglia, which are extremely slender and form a very fine network. In the peripheral parts, on the other hand, the tissue is less dense, the cells of the arachniform neuroglia are much larger and the intertwinement is looser. The nerve-cells although more scarce than normal are more numerous and less altered than in the central parts. The differential diagnosis, anatomically, between this and other forms of idiocy is fully pointed out, and an anatomical comparison with other pathological processes of the central nervous system, but unaccompanied by idiocy, such as epilepsy, multiple sclerosis, syphilitic cerebral lesions, and cerebral tumours, is made. This part of the book contains the best description of the affection, which is well known to the reviewer, that has yet been published.

In the second part the author refers to and describes the classifications of idiocy adopted by Bourneville, Ireland, Shuttleworth and the reviewer, Hammarburg and Tanzi, and proposes a classification of his own. He divides idiocy into three great classes; first, that which is due to defect of development of the brain, or idiocy of endogenous origin; secondly, that which is caused by pathological processes of the brain and its convolutions and by accident, or idiocy of exogenous origin; and thirdly, that which is due to mixed causes. These three classes with their groups and subdivisions are presented in the form of a table.

In the third part, Dr. Pellizzi quotes the opinions of the ancient and modern writers who have written on epileptic idiocy, and goes on to say that he recognises two chief forms of it, the first being idiocy with essential epilepsy, and the second idiocy with idiopathic epilepsy. He also mentions the existence of a primitive, true, endogenous idiocy with epilepsy, epileptic dementia and epileptic idiocy.

A very full bibliography is given at the end of the book, which is made more interesting by some excellent engravings, which illustrate the macroscopical and microscopical appearances which have been observed by the author in tuberose sclerosis.

The book contains much interesting information which will be useful to specialists in mental and nervous diseases, and especially to those who have to treat idiocy and imbecility.

FLETCHER BEACH.

An Atlas of the Medulla and Mid-brain. By FLORENCE R. SABIN, M.D. Edited by Dr. Henry Knower. (Baltimore, 1901.)

THIS Atlas, described as an illustrated laboratory manual, is planned to meet the wants of the student working at practical neurology, and to afford him the means of accurately and satisfactorily studying structures by quickly comparing his own sections with the figures of the Atlas. A point of value which is observed on looking through the Atlas, and which is less commonly noticed in illustrated manuals, is the number of longitudinal and horizontal sections through the ponto-medullary region. The illustrations consist of a series of sections passing through the medulla, pons and mid-brain of a new-born infant, in the first series from the dorsal to the ventral surface, and in the second from the spinal cord upwards towards the cerebrum. Eight full-page plates at the end of the Atlas illustrate portions of Miss Sabin's well-known model of the mid and hind-brain. The Atlas will no doubt be of assistance to those requiring a laboratory manual.

W. A. TURNER.

Beiträge zur Pathogenese und pathologischen Anatomie der Epilepsie. von Dr. L. W. WEBER. (Jena, 1901.)

THIS is a short memoir upon the pathological appearances, coarse and microscopic, found in a series of thirty-five cases of true epilepsy. The results obtained by the author may be briefly stated: in many cases there is a thickening of the cortical neuroglia, partly from an increase in the number of glia corpuscles and partly from the presence of a fine network of glial fibres, the thickness and extent of which is directly proportional to the duration of the disease.

The changes in the blood-vessels of the cerebral cortex consist, in acute and recent cases, of hyperæmia, œdema of the lymph and perivascular spaces, extravasations and small-celled infiltrations; in chronic cases, of thickening of the arterial walls, and the formation of new blood-vessels in the superficial cortical layers; while in the pyramidal cells Nissl changes may be detected in old cases, pigment deposits, shrinkage of the cell bodies and falling off of the processes.

The general bearing of these observations upon the clinical types of the disease is that in congenital and early cases there is found chiefly the subpial fibrous network already mentioned, sclerosis of the blood-vessels, and disappearance of the nerve elements; while in the so-called "late epilepsy" the chief changes are irregular thickening of the cortical neuroglia with disturbance of the normal cortical structure.

There is nothing in these alterations which may not be described as consequences of epilepsy.

W. A. TURNER.

Der Farbensinn der Tiere. By W. A. NAGEL. Wiesbaden. (J. F. Bergmann, 1901, pp. 32.)

IN this lecture Professor Nagel discusses the existence and nature of the colour sense in animals. He regards protective colouration as indicating that most of the higher animals have a colour sense, but points out that zoologists are usually content to assume the existence of this sense, and do not seem to realise that the assumption is one capable of verification or disproof by exact methods.

Professor Nagel points out that one difficulty in the exact investigation of the colour sense in animals has only become apparent owing to recent advances in our knowledge of retinal physiology. In the earlier work on the subject the factor of brightness was neglected, and in reading early accounts one has no means of telling how far the reaction of an animal to a given colour was due to the colour-tone and how far to the brightness of the colour. In more recent investigations attention has been paid to this factor, but one point has been lost sight of. We know that the relative brightness of two colours may be very different to a colour-blind man and to an individual with normal colour sense. Further, the two colours may vary greatly in relative brightness for the same individual under different conditions of retinal adaptation. The possibility is suggested that the relative brightness of two colours may not be the same to different animals, and that an animal appearing to distinguish two colours, which are of the same brightness to the normal human eye, may nevertheless be guided by differences of brightness and not by differences of colour tone.

As a matter of fact, Abelsdorff has found that there is a marked difference between the pigeon and owl in the amount of constriction of the pupil produced by the action of red and blue rays, and since the amount of constriction of the pupil is, so far as we know, proportional to the brightness of a colour, we may conclude, with a considerable degree of probability, that the relative brightness of red and blue is not the same to the two birds.

Another objective measure of brightness is the action current. Nagel and Himstedt have experimented with this on the frog's eyeball, in the conditions of adaptation both to light and dark. In the former condition the largest variation was found on stimulation with yellow light from about the line "D," while the dark-adapted eyeball reacted most to green light from near the thallium line. That is to say, the maxima of action for the frog's retina in light and dark adaptation exactly corresponded with the places of maximum brightness of the spectrum as seen by the human eye in light and dark adaptation respectively. There is much reason to believe that the shifting of the place of maximum brightness is due to the different degree of sensibility of the rods and cones in the two conditions of adaptation, and it is therefore interesting to find the same shifting in the frog, whose retina also contains both rods and cones.

Nagel has also attempted to investigate the action currents in pigeons and fowls (which have few rods and no visual purple), but up to the present his efforts have been unsuccessful owing to the rapid changes which occur after enucleation.

Nagel's observations do not, of course, prove that the colour system of the frog is the same as that of man, nor do they even show that this animal has a colour sense at all, but they increase the probability of these assumptions and they show that in future experimental work on the subject one may reasonably assume that colours which are of equal brightness to the human eye will have an equivalent action, so far as brightness is concerned, on the frog's eye.

W. H. R. Rivers.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

At a Clinical Meeting held at 11, Chandos Street, on Thursday, October 31, at 8.30 p.m., the following cases were shown:—

Dr. Ferrier.—*Case of successful removal of a cerebellar tumour.*

The patient shown, Walter B., aged 12, was admitted into King's College Hospital on May 22, 1900.

He had been suffering for several months previously with headache and vomiting—symptoms which had been increasing in severity for two months previous to admission.

He had a cerebellar gait and there was well-marked double optic neuritis. The right occiput was somewhat more prominent than the left, and pressure over this region caused some tenderness. This swelling was supposed to have originated from a fall downstairs in infancy.

It was decided to trephine over this region, and the operation was performed by Mr. Burghard on June 13. The bone was found to be unusually thin, and the right lobe of the cerebellum on exposure was seen to bulge considerably, but no tumour was visible. On the lateral lobe being incised a tumour was disclosed apparently filling up the whole of the interior of the lobe. This was readily shelled out with the finger, and a tongue-like process, which extended more deeply, was cut off together with some doubtful-looking tissue in which it appeared to end. Great collapse (from hæmorrhage) followed the operation, but after transfusion of saline solution and the administration of stimulants the pulse improved and the patient made an uninterrupted recovery. The optic neuritis gradually subsided and the patient left the hospital on July 22 quite well. He has continued so since, and attends school and engages in all outdoor games like any other schoolboy.

The tumour was found on microscopical examination to have the characters of a fibro-endothelioma.

Dr. FERRIER and Dr. ALDREN TURNER.—*Case of successful removal of a cerebral tumour.*

Mrs. P., aged 34, began to have twitchings affecting the left side of the face in 1894, which continued with increasing frequency and severity into 1895, when she had two fits which affected the left limbs as well as the face. During 1898 the seizures were of frequent occurrence. In March, 1900, left hemiplegia made its appearance, and in January, 1901, optic neuritis, the fits meanwhile continuing, some of them having the character of general epileptic seizures.

She was admitted into King's College Hospital, under Dr. Ferrier's care, on July 15, 1901. On August 10 Mr. Cheatle operated and removed a small fibrous, encapsulated tumour from the lower part of the right Rolandic region.

The patient has made an uninterrupted recovery, with entire cessation of fits, disappearance of the hemiplegia and resolution of the optic neuritis.

Microscopically the tumour was found to be a fibroma.

Dr. ROBERT HUTCHISON. — *Unilateral inco-ordination, with inability to rotate the eyes downwards, in a mentally defective boy.*

Thomas M., aged 6 years 1 month, was brought to hospital for inability to walk properly. He has never walked well, but for the last six months the difficulty has been increasing, and for the same length of time it has been noticed that his head tended to be bent over towards the left shoulder. There is no history of any definite illness, but he has never been intelligent nor able to use more than one or two words; he has been well cared for but has never gone to school. There is no history of fits.

Patient is the youngest of the family. There are four brothers and two sisters alive and healthy, but two miscarriages immediately preceded the birth of this child.

Mother died, aged 42 years, after a week's illness, beginning with a fit. The father is alive and well. There is no history of syphilis. One maternal uncle died of general paralysis of the insane, and his son is at present in an asylum with the same disease.

Patient is well developed for his years, but has a vacant expression and is evidently an imbecile. He has a slight alternating squint, and the lower part of the sclerotic in the right eye is exposed from an upward inclination of the globe. He tends to keep his head a little flexed laterally to the left but

without rotation, but it can be restored to the vertical position without difficulty. He presents no evidence of congenital syphilis. Muscular power in the right limbs is normal; the grasp on the left side is feeble and he often uses the right hand to raise the left arm. There is marked intention tremor with wide excursus on attempted use of the left arm. He walks with rather a wide base, but without deflection; the left leg in walking is raised high with a jerky motion, the foot thrown forward and the heel brought down sharply.

Both knee-jerks are +, especially the left; ankle-clonus not obtained; extensor response on left, flexor on right side. No tendon reflexes obtained in the upper limbs. He has complete control of his sphincters. The pupils are equal and react normally. He cannot be got to rotate the eyes downwards, and in order to see objects below the horizontal plane he bends his head forwards. The other ocular muscles are normal, but he has occasionally slight lateral nystagmus. There is no optic neuritis, and no apparent paralysis of other cranial nerves. There is no spinal curvature, and the other organs are healthy.

Dr. BEEVOR.—*A case of muscular atrophy with loss of sensation over the 1st dorsal root area.*

Ethel C., aged 19, quite well till three years ago. When 12 years old had scarfet fever; 13 years had chorea; 15 years had second attack, after which she began to have wasting of the right hand; no pain.

At present has atrophy of all the right hand muscles, with slight weakness of the flexor of fingers. Electric Reaction.—Thenar muscles do not react to faradism, interossei and hypothenar react feebly. She has a patch of anæsthesia and analgesia over area of right dorsal root.

Dr. BATTEN.—*Spasmodic movements of the limbs in an infant.*

W. M., male, aged 1½ years old, the fifth child of a family of five, of whom all the others are healthy, was born at full time in natural labour.

Was quite well till 9 months old and was able to sit up. When 9 months old had a series of fits, and since then the child has been weaker and the spasmodic movements have developed.

The child is well-nourished, is on its back and has a continuous series of irregular clonic movements of the head, arms, legs, trunk and fingers. The legs in particular are frequently

suddenly flexed on the trunk, and only gradually return to their normal position. The mental condition is probably defective, there is no rigidity. Knee-jerks equal and brisk. No ankle-clonus. Plantar gives flexor response. Vision apparently good.

Dr. HEAD.—(1) *A young woman with muscular atrophy, probably of neuritic origin.*

Emily B., aged 27, single, a confectionery packer.

With the exception of several attacks of anæmia she was well until Christmas, 1899. She then noticed that her right thumb "gave in" at her work, and that the first dorsal interosseous space was wasted. She attended St. Bartholomew's Hospital during the winter of 1900, and the London Hospital since February, 1901. During the last twelve months the left hand has begun to waste.

Before the wasting in the hands began she had pains in the arms and shoulders, and she still has occasional pains which are not severe.

She now shows marked wasting of the right hand, particularly of the thenar and hypothenar group and interossei, particularly the first dorsal interosseus. In the left hand the first dorsal interosseus is wasted and the thenar muscles are smaller than normal.

On the right side the supra- and infraspinatus and the deltoid are distinctly wasted.

All the muscles of the right fore-quarter can be made to respond to faradism except the extremely wasted muscles on the outer side of the thumb; the deltoid responds feebly. To galvanism the deltoid shows a slow contraction and A.C.C. > K.C.C. In the thenar muscles there is no actual reversal, but contraction is slow. In the first dorsal interosseus A.C.C. > K.C.C.

All the muscles of the left fore-quarter react normally to faradism, but those of the thenar eminence do not respond so easily as the hypothenar muscles. To galvanism all muscles react normally, but contraction is slow in the first dorsal interosseus and the muscles of the thumb.

There is no paræsthesia or anæsthesia.

Reflexes, cranial nerve, and sphincters are normal.

Improvement has taken place under massage.

(2) *A Case for diagnosis.*

Ellis B., aged 61, a brushmaker, was admitted to the London Hospital under Mr. Roxburgh on account of pain in the left knee.

Thirty-four years before this knee had been cut with a knife, had suppurated and ankylosed in a semiflexed position. About May, 1900, the pain, which had always been present to a bearable degree, became intolerable.

All attempts to relieve this pain were ineffectual, and a Stokes-Gritti amputation was performed in October, 1900, by Mr. Barnard.

All pain ceased and he says he was never so comfortable in his life—able to do his work and to get about on crutches.

On December 22, 1900, he spent the day in the Hospital for the Christmas Entertainment. At the end of the day whilst standing he suddenly pitched forwards but did not lose his senses. His foot was found to be completely paralysed.

No history of syphilis can be obtained, and except for the ankylosed knee he has been healthy.

The left leg has been amputated and is now free from pain. Movement and muscular development of the thigh and hip good. Sensation unaffected.

All movements of the right foot are absent. He cannot move the toes or dorsiflex and plantar-extend the foot. The knee is slightly flexed, possibly from constantly sitting in a chair. Faradism evokes no response in the tibialis anticus, extensor longus digitorum, peroneus longus and soleus. With galvanism the contraction is sluggish and in the soleus A.C.C. > K.C.C.

Pain, heat and cold are not appreciated over an area that appears to be that of the first sacral.

Touch is lost over the same area, but the edges are less defined.

The knee-jerk cannot be obtained in the right knee.

Pupils react to light and accommodation.

Sphincters act normally.

Dr. MUSKENS, of the Hague, Holland, gave a lantern demonstration illustrating *Disturbances of sensation of segmental distribution in Tabetics and Epileptics*. In order to record the results as accurately as possible the patients were photographed both front and back, using different bony points as landmarks, having first examined and marked out on the skin the analgesic areas (with vertical lines) and the anæsthetic areas with horizontal lines.

Examining in this way carefully every individual case of tabes the records confirmed the view that the disturbances of sensation in locomotor ataxia really developed in segmental areas. In incipient cases he succeeded frequently in marking out, as the

upper border of the thoracic area, the upper margin of the first dorsal root area quite accurately. The lumbar analgesic (or hypalgesic) area appeared not to be limited to one particular segment in incipient cases with the same regularity (photographs shown). In other incipient cases the different stages of anæsthesia had been followed out and information obtained about the way in which different forms of defective æsthesia follow upon each other. (Compare L. J. J. Muskens, "On the development of the Disturbances of Sensation, &c.," *British Medical Journal*, December 9, 1899.) In the rare cases in which there was an opportunity to examine the cord of such cases, the corresponding anatomical substratum could best be demonstrated by counting (Weigert-stain) the relative number of degenerate fibres in the posterior columns of the different spinal segments.

Subsequently, having an opportunity to examine a great number of epileptics, Dr. Muskens was struck with the fact that in a great number of these the same areas on the trunk, which were so frequently found analgesic in tabetics, presented a marked hypalgesia for pinpricks. This lack of acute sensation for pain was particularly striking in several cases which were examined a short while before a fit came on. The borders of the hypalgesic areas showed mostly a marked segmental character. A number of cases were then examined every day for a considerable period. In most cases the extent of the hypalgesic field was found to vary a great deal. In most cases where fits seldom occurred the influence of the discharge upon the extent of the hypalgesic area became very apparent. In some of these cases the discharge could be predicted with some probability for one or more days. Diagrammatic curves, demonstrating the variation in the extent and intensity of the hypalgesia, showed in many cases a marked increase in the amount of hypalgesia shortly before the fit, even up to complete and total analgesia of the body; on the other hand, some time after a fit, or a series of fits, was over, general hypalgesia was usually present over the entire skin. Generally, also, in the intervals between the discharges the curves recording the upper and lower segmental border showed a rather irregular increase and decrease of the extent of the hypalgesic area. The return of the sensibility to pain of both sides of the body appeared to be different in a number of cases; the side which discharged first and strongest, as a rule, regained its sensation last.

Dr. Muskens did not think that these hypalgesic segmental areas could be looked upon as characteristic of epileptics, although in only a few non-epileptics a similar hypalgesic area was found.

Some sensory areas showed a remarkable tenacity in regard to their sensation to pain, even in otherwise very complete pre-epileptic analgesia, and also regained their normal sensation sooner than the neighbouring parts, viz., a small field, practically coinciding with the second branch of the fifth nerve, one on the inner side of the ball of the thumb, and one occupying part of the planta pedis. These same areas appear to show a similar tenacity in regard to the sensation to pain in apoplectic and other forms of cerebral hemianalgesia.

The Meeting held at 11, Chandos Street on Thursday, December 5, at 8.30 p.m., was devoted to the consideration of Methods of Psycho-physical Research.

Dr. RIVERS explained:—(1) *Methods of estimating mental fatigue and practice.*

This subject was illustrated chiefly by an account of the results of a comparative investigation on Papuan and English children. Work for three-quarters of an hour was carried on at intervals of a day, two days, a week and a month. The influence of pauses of five minutes and a quarter of an hour during the period of work was also shown.

The full account will be published in the Reports of the Cambridge Expedition to Torres Straits.

(2) *Methods of measuring visual illusion.*

This subject was also illustrated by means of comparative work on Papuan and English observers.

Dr. MOTT and Dr. W. G. SMITH demonstrated a membrane key and a falling shutter.

The Index of BRAIN for the Volumes I. to XXIII. inclusive, that is, from its commencement to the end of 1900, is now ready.

Members of the Neurological Society can obtain the volume from Messrs. MACMILLAN & Co., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Society the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

As the cost of the Index has been considerable the Council hope that every member of the Society will order at least one copy.

EDITOR.

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Rules

AND

LIST OF OFFICE BEARERS AND MEMBERS

OF THE

Neurological Society of London,

1901

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1900.

LONDON:
JOHN BALE, SONS & DANIELSSON, LTD.
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Neurological Society of London.

REPORT OF COUNCIL.

JANUARY 24, 1901.

The Council has much pleasure in reporting the continued success and prosperity of the Society. The members now number 215.

The Inaugural Address was delivered at the Annual General Meeting, on February 8th, 1900, by Dr. AUGUSTUS WALLER, F.R.S., "On the Excitability of Nervous Matter, with especial reference to the Retina."

The greatest feature in the year's programme has been the delivery of the second Hughlings Jackson Lecture, on Thursday, November 29th, 1900, by Professor E. HITZIG, of Halle, who chose for the subject of the lecture, "Hughlings Jackson and the Cortical Motor Centres in the Light of Physiological Research."

It is with great regret that the Council announces that Dr. A. de WATTEVILLE has resigned the Editorship of "Brain." When accepting his resignation the following resolution was unanimously adopted: "The Council accepts with great regret Dr. de Watteville's resignation of the Editorship of 'Brain,' and desires to take this opportunity of recording the deep debt of gratitude that the Society owes him for the way in which he has conducted the Journal for the past twenty years. The Council feels that parting with Dr. de Watteville is an event of great moment to the Society, for he has not only brought 'Brain' to a high standard of perfection and secured for it a great European reputation, but even the existence of the Journal at the present time is due to his energetic action at a critical juncture in 1880. Moreover, the Council is mindful that the Society itself took origin on Dr. de Watteville's initiative, at a meeting held at his house, on November 14th, 1885."

The Council regrets the loss that the Society has sustained by the death of a distinguished member, Professor Sir THOMAS GRAINGER STEWART, of Edinburgh, and of Mr. A. SYMONS ECCLES.

The Council has also to report with much regret the resignation of the Senior Secretary, Dr. RISIEN RUSSELL, after four years of valuable service rendered to the Society.

An abstract of the Proceedings of the Society appended to this Report shows the character and value of the work of the Society during the past year.

Proceedings of the Society during the year 1900.

February 8th, Dr. A. WALLER, F.R.S., Inaugural Address, "The Excitability of Nervous Matter, with especial reference to the Retina."

March 15th, Dr. F. E. BATTEN, "Case of Congenital Cerebellar Defect." Dr. FARQUHAR BUZZARD, "A Case of Cerebral Diplegia." Dr. RISIEN RUSSELL, (1) "Two Cases of Thomsen's Disease (Myotonia Congenita)"; (2) "A Case of Functional Wry-neck, with Clonic Spasm of the Palate." Dr. EDWIN BRAMWELL, "Two Cases of Myasthenia Gravis." Dr. JAMES COLLIER, "A Case of Muscular Atrophy in the Upper Extremities."

May 10th, Dr. WAKELIN BARRATT, "Subdural Blood Cyst compressing Right Hemisphere." Dr. F. E. BATTEN, (1) "Series of mounted Specimens of some of the Grosser Pathological Changes found in the Nervous System"; (2) "A Cyst in the Seventh Left Dorsal Posterior Root Ganglion, from a Case in which a Subcutaneous Hæmorrhage had taken place in the Distribution of the Seventh Left Thoracic Root some months previously." Dr. JAMES TAYLOR, "Sections of the Nuclei of the Third and Sixth Cranial Nerves in a Case of Ophthalmoplegia Externa." Dr. J. S. COLLIER, (1) "Photographs and Bone of a Case of Leontiasis Ossea"; (2) "Skull from a Case of Syringomyelia simulating Leontiasis Ossea"; (3) "Endothelioma of Skull." Dr. A. BRUCE, "Tumour of Third Ventricle."

June 21st, Dr. HEAD, "Two Cases of Muscular Atrophy in Young Women, probably of Neuritic Origin." Dr. COLMAN, "A Case of 'Ophthalmoplegic Migraine.'" Dr. LEES, (1) "Hæmatomyelia"; (2) "Hysterical Contracture." Dr. LEONARD GUTHRIE, "Thomsen's Disease." Dr. NEWTON PITT, (1) "Insular Sclerosis"; (2) "Two Cases of Doubtful Nature, possibly Insular Sclerosis."

October 25th, in the Physiological Laboratory, St. Mary's Hospital, Mr. VICTOR HORSLEY, F.R.S., (1) "Note on the Course of the Fillet Fibres in the Cat"; (2) "Further Note on a Cat in which Brown-Séquard Fits were obtained after a Lesion of the Medulla Oblongata, the Fits being still obtained after Complete Division of the Mesencephalon." The President, "The Excitability of Nervous Matter- its Last Sign of Life."

November 29th, the Second Hughlings Jackson Lecture, Professor EDUARD HITZIG, of Halle, "Hughlings Jackson and the Cortical Motor Centres in the Light of Physiological Research."

BALANCE SHEET, 1900.

Cr. NEUROLOGICAL SOCIETY. **Dr.**

Receipts.	£	s.	d.
To Balance	45	9	2
Subscriptions	230	3	0
Half Share of profit on "Brain" ...	34	19	4
Contribution by Messrs. Macmillan towards cost of Illustrations in "Brain"	11	13	0
	£322	4	6

Expenditure.	£	s.	d.
By Stationery and Printing—Bale & Danielsson	27	4	8
Illustrations—Bale & Danielsson ...	76	0	0
Microscopes and Lanterns—			
C. Baker £7 2 0			
A. Hawes 2 4 6			
	9	6	6
Hire of Rooms and Refreshments—Medical Society	20	15	0
Messrs. Macmillan for publishing "Brain"	121	0	0
Bank Charges	0	4	0
Petty Cash	1	13	0
Balance	66	1	4
	£322	4	6

Audited and found correct December 5, 1900,

CHARLES E. BEEVOR, *Hon. Treas.*

H. G. TURNEY,
FREDERICK E. BATTEN.

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorar Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due, shall be considered as equivalent to resignation.

15.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

16.—Five Members of the Council shall form a quorum.

17.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

18.—The office of Vice-President shall be tenable for two years one Vice-President being elected every year.

19.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

20.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

21.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

22.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

23.—There shall be six Ordinary Meetings annually, of which the first held in each year shall be the Annual General Meeting.

24.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

25.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

26.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

27.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

28.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

29.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by R. Percy Smith, M.D. F.R.C.P.

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Professor Wundt, Leipzig.
,, Hitzig, Halle.
Sir Samuel Wilks, Bart., London.

CORRESPONDING MEMBERS.

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,, Golgi, Pavia.
,, Goltz, Strasburg.
, Grasset, Montpellier.
,, Jolly, Berlin.
,, Luciani, Rome.
,, Munk, Berlin.
,, Obersteiner, Vienna.
,, Retzius, Gustaf, Stockholm.
,, Ribot, Paris.
Dr. Weir Mitchell, Philadelphia.

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

1886 J. Hughlings Jackson, M.D., F.R.S.
1887 Sir Samuel Wilks, Bart., M.D., F.R.S.
1888 Sir James Crichton-Browne, M.D., F.R.S.
1889 Jonathan Hutchinson, F.R.S.
1890 Thomas Buzzard, M.D.
1891 John S. Bristowe, M.D., F.R.S.
1892 H. Charlton Bastian, M.D., F.R.S.
1893 Edward A. Schäfer, F.R.S.
1894 David Ferrier, M.D., F.R.S.
1895 Sir William H. Broadbent, Bart., M.D., F.R.
1896 Alexander Hill, M.A., M.D.
1897 George H. Savage, M.D.
1898 Victor Horsley, M.B., F.R.C.S., F.R.S.
1899 W. H. Gaskell, M.D., F.R.S.
1900 Augustus Waller, M.D., F.R.S.
1901 W. Julius Mickle, M.D.

LIST OF HUGHLINGS JACKSON LECTURERS.

1897 J. Hughlings Jackson, M.D., F.R.S.
1900 Professor Eduard Hitzig, Halle.

*** Members are requested to communicate with the Secretaries when corrections are necessary.

—:o:—

EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.	Tr., Treasurer.
Pres., President.	Sec., Secretary.
V.P., Vice-President.	C., Member of Council.

H.J.L., Hughlings Jackson Lecturer.

ORDINARY MEMBERS.

Elected

1899 ABRAHAMS, BERTRAM, M.B., M.R.C.P., 14, Welbeck Street, W.

1892 ALEXANDER, ROBERT REID, M.D., The Asylum, Hanwell, W.

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1894 ANDRIEZEN, W. LLOYD, M.D., 7, Apsley Terrace, Acton, W.

1891 BALLANCE, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897-1900.)

O.M. BARLOW, Sir THOMAS, Bart., M.D., F.R.C.P., 10, Wimpole Street, W.

1897 BARRATT, J. O. WAKELIN, M.D., F.R.C.S., Pathological Laboratory, West Riding Asylum, Wakefield.

O.M. BASTIAN, H. CHARLTON, M.D., F.R.C.P., F.R.S., 8A, Manchester Square, W. (Pres., 1892; V.-P., 1891; C., 1886-89, 1893-95.)

1897 BATTEN, FREDERICK E., M.D., M.R.C.P., 124, Harley Street, W.

O.M. BEACH, FLETCHER, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey.

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1888 BIRT, ERNEST, M.D., West Riding Asylum, Wakefield.

1898 BOLTON, JOSEPH SHAW, M.D., B.S., B.Sc., Claybury Hall, Woodford, Essex.

1896 BOND, C. HUBERT, M.D., D.Sc., London County Asylum, Bexley, Kent.

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,, BRADBURY, JOHN B., M.D., F.R.C.P., 4, St. Peter's Terrace, Cambridge.

1889 BRADFORD, J. R., M.D., D.Sc., F.R.C.P., F.R.S., 8, Manchester Square, W.

O.M. BRAMWELL, BYROM, M.D., F.R.C.P.Ed., 23 Drumsheugh Gardens, Edinburgh. (C., 1894

Elected

1899 BRAMWELL, EDWIN, M.B., C.M., M.R.C.P., 2, Randolph Place, Edinburgh.

1895 BRAMWELL, JOHN MILNE, M.B., C.M., 2, Henrietta Street, W.

1892 BRISTOWE, HUBERT CARPENTER, M.D., Wrington, Somerset.

O.M. BROADBENT, Sir WM. H., Bart., M.D., F.R.C.P., F.R.S., 84, Brook Street, W. (Pres., 1895; V.-P., 1893; C., 1886-87, 1896.)

1894 BRODIE, T. GREGOR, M.D., 55, Marlborough Mansions, West Hampstead, N.W.

1897 BROWN, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.

1888 BROWN, SANGER, M.D., 100, State Street, Chicago.

O.M. BRUCE, ALEX., M.D., F.R.C.P.Ed., 8, Ainslie Place, Edinburgh. (C., 1895.)

,, BRUCE, J. MITCHELL, M.D., F.R.C.P., 23, Harley Street, W.

1898 BRUCE, SAMUEL NOBLE, M.R.C.S., 15, Queensboro' Terrace, Hyde Park, W.

O.M. BRUNTON, Sir T. LAUDER, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896-98.)

1895 BRYAN, FREDERICK, M.B., Horton Manor Asylum, Banstead Downs, Surrey.

,, BRYANT, JOHN HENRY, M.D., M.R.C.P., 8, Mansfield Street, Portland Place, W.

1890 BURY, JUDSON SYKES, M.D., F.R.C.P., 10, St. John Street, Manchester.

1900 BUZZARD, E. FARQUHAR, M.B., M.R.C.P., 137, Bedford Court Mansions, W.C.

O.M. BUZZARD, THOMAS, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.P., 1888-89; C., 1886-87, 1891-95.)

1898 CALEY, H. A., M.D., F.R.C.P., 24, Upper Berkeley Street, W.

,, CAMPBELL, HARRY, M.D., F.R.C.P., 23, Wimpole Street, W.

1887 CATTELL, J. MCK., M.A., Ph.D., Columbia College, New York, U.S.A.

1899 CHAMBERS, JAMES, M.A., M.D., The Priory, Roehampton.

1891 CLARKE, J. MICHELL, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol.

1898 CLINCH, THOMAS ALDOUS, M.D., 16, Regent Street, Nottingham.

1892 COLE, ROBERT HENRY, M.D., M.R.C.P., 48, Upper Berkeley Street, Portman Square.

1899 COLLIER, J. S., M.D., B.Sc., M.R.C.P., 57A, Wimpole Street, W.

1890 COLMAN, W. S., M.D., F.R.C.P., 22, Wimpole Street, W. (Sec., 1899-1901.)

,, CORNER, HARRY, M.D., Brook House, Southgate, Middlesex.

1899 COUPLAND, SIDNEY, M.D., F.R.C.P., 16, Queen Anne Street, W.

1895 DAVIDSON, ANDREW, M.D., The County Asylum, Dorchester, Dorset.

1897 DAWSON, WILLIAM, R., M.D., F.R.C.P.I., Farnham House, Finglas, Co. Dublin, Ireland.

Elected

1894 DEAN, H. PERCY, M.S., F.R.C.S., 69, Harley Street, W.

1898 DODD, HENRY WORK, F.R.C.S., 136, Harley Street, W.

O.M. DRESCHFELD, JULIUS, M.D., F.R.C.P., 3, St. Peter's Square, Manchester. (V.P., 1898-99.)

,, DRUMMOND, DAVID, M.D., 6, Saville Place, Newcastle-on-Tyne.

1892 DUPUY, EUGENE, M.D., 53, Avenue Montaigne, Paris.

1899 FAWCETT, JOHN, M.D., M.R.C.P., 24, St. Thomas Street, S.E.

O.M. FERRIER, DAVID, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W., (Pres., 1894; V.P., 1892-3; C., 1886-90-95.)

,, FOX, E. LONG, M.D., F.R.C.P., Church House, Clifton, Bristol.

,, GASKELL, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (Pres., 1899; V.P., 1897-98; C., 1896, 1900.)

,, GEE, SAMUEL J., M.D., F.R.C.P., 31, Upper Brook Street, W.

,, GODLEE, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1899 GOODALL, EDWIN, M.D., M.R.C.P., Joint Counties Asylum, Carmarthen.

1898 GOODLIFFE, JOHN HENRY, M.D. Aberd., Norton Hall, near Sheffield.

1897 GOSSAGE, ALFRED MILNE, M.B., M.R.C.P., 54, Upper Berkeley Street, W.

1892 GOTCH, FRANCIS, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 GRANT, J. DUNDAS, M.D., F.R.C.S., 18, Cavendish Square, W.

O.M. GUNN, R. MARCUS, M.B., F.R.C.S., 54, Queen Anne Street, W. (C., 1899-1900.)

1895 GUTHRIE, LEONARD, M.D., F.R.C.P., 15, Upper Berkeley Street, W.

1893 HALLIBURTON, WILLIAM DOBINSON, M.D., F.R.C.P, F.R.S., Church Cottage, 17, Marylebone Road, N.W.

O.M. HAMILTON, D. J., M.B., F.R.S.E., University, Aberdeen.

1898 HANBURY, WILLIAM R., M.R.C.S., L.R.C.P., County Asylum, Brentwood, Essex.

1897 HARRIS, WILFRED J., M.D., M.R.C.P., 19, Oxford Terrace, W.

1898 HARTLEY, W. DARLEY, M.D., M.R.C.P., Park House, Kloof Street, Cape Town, South Africa.

,, HASLETT, JOHN HANFIELD, M.R.C.S., L.R.C.P, Halliford House, Sunbury-on-Thames.

1891 HAWKINS, HERBERT PENNELL, M.D., F.R.C.P., 56, Portland Place, W.

O.M. HAYCRAFT, J. B., M.D., D.Sc., F.R.S.E., 1, St. Andrew's Place, Cardiff.

1891 HEAD, HENRY, M.D., F.R.C.P., F.R.S., 61, Wimpole Street, W. (C., 1900, Sec. 1901.)

O.M. HILL, ALEX., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.P., 1895; C., 1892-94.)

Elected

O.M. HORSLEY, VICTOR, F.R.C.S., F.R.S., 25, Cavendish Square, W. Pres., 1898; V.P., 1896-97; C., 1892-93, 1899.)

1889 HUGGARD, WILLIAM R., M.D., F.R.C.P., Davoz Platz, Switzerland.

1896 HULME, GEORGE FREDERICK, M.B., C.M., South View, Montague Road, Felixtowe.

1889 HUMPHRY, LAURENCE, M.D., M.R.C.P., Lensfield, Cambridge.

O.M. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.P., 1887-88; C., 1886, 1890.)

1889 HYSLOP, T. B., M.D., C.M., Bethlem Royal Hospital, Lambeth, S.E.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.C.P., F.R.S., 3 Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887, 1892.)

,, JESSOP, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 JOHNSTON, GEORGE, M.D., M.R.C.P., 3, Montagu Place, Montagu Square, W.

1892 JONES, HENRY LEWIS, M.D., F.R.C.P., 61, Wimpole Street, W.

1894 JONES, ROBERT, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

,, KIDD, PERCY, M.D., F.R.C.P., 60, Brook Street, W.

1896 LANGDON, F. W., M.D., Cincinnati, Ohio, U.S.A.

1898 LANGDON-DOWN, REGINALD L., M.B., M.R.C.P., 81, Harley Street, W.

O.M. LANGLEY, J. N., M.A., D.Sc., F.R.S., Trinity College, Cambridge. (V.P., 1901; C., 1898-99.)

1888 LAWFORD, J. B., M.D., F.R.C.S., 99, Harley Street, W.

O.M. LEES, D. B., M.D., F.R.C.P., 22, Weymouth Street, W. (C., 1900, 1901.)

1897 LEWIS, H. WOLSELEY, M.R.C.S., L.R.C.P., The Manor Asylum, Horton, Epsom.

1899 LORD, JOHN R., M.B., C.M., London County Asylum, Bexley, Kent.

1897 LOEWENTHAL, MAX, M.D., M.R.C.P., 126, Princes' Road, Liverpool.

1898 MACCORMAC, JOHN M'GEE, M.D., 31, Victoria Place, Belfast.

,, MCALDOWIE, ALEXANDER M., M.D., 6, Brook Street, Stoke-on-Trent.

1891 MACDONALD, PETER WILLIAM, M.D., C.M., The County Asylum, Dorchester, Dorset.

,, MACKENZIE, HECTOR WILLIAM GAVIN, M.D., F.R.C.P., 34, Upper Brook Street, Grosvenor Square, W.

O.M. MACKENZIE, STEPHEN, M.D., F.R.C.P., 18, Cavendish Square, W. (C., 1896-98.)

1896 MACKINTOSH, ASHLEY W., M.A., M.D., 9, Bon Accord Square, Aberdeen.

1899 MACLULICH, PEERS, M.D., B.Ch., Joint Counties Asylum, Carmarthen.

1889 MACPHAIL, S. R., M.D., Borough Asylum, Rowditch, Derby.

Elected

O.M. MAGUIRE, ROBERT, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)

,, MANN, J. DIXON, M.D., F.R.C.P., 16, St. John's Street, Manchester.

1894 MARTIN, SIDNEY, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.

1891 MAY, W. PAGE, M.D., M.R.C.P., Helouan, Cairo, Egypt.

1890 MENZIES, WILLIAM FRANCIS, M.D., B.Sc., M.R.C.P., Cheddleton Asylum, Leek, Staffordshire.

O.M. MERCIER, C., M.B., M.R.C.P., Flower House, Southend, Catford, S.E. (C., 1899-1901.)

1897 MEYER, ADOLF, M.D., Clark University, Worcester, Mass., U.S.A.

O.M. MICKLE, W. JULIUS, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (Pres., 1901; V P., 1900; C., 1895-97.)

1895 MOORE, NORMAN, M.D., F.R.C.P., 94, Gloucester Place, W.

O.M. MORRIS, MALCOLM, F.R.C.S.Ed., 8, Harley Street, W.

1888 MOTT, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894-98; C., 1899-1901.)

1899 MOULD, GILBERT E., M.R.C.S., L.R.C.P., The Grange, nr. Rotherham.

1894 MURRAY, GEORGE R., M.A., M.D., F.R.C.P., 11, Ellison Place, Newcastle-on-Tyne.

1889 MURRAY, H. MONTAGUE, M.D., F.R.C.P., 25, Manchester Square, W.

O.M. NETTLESHIP, EDWARD, F.R.C.S., 5, Wimpole Street, W.

,, NICOLSON, DAVID, C.B., M.D., M.R.C.P.Ed., Balgownie, Guildford, Surrey.

1888 NIERMEYER, J. H. H., M.D., Amsterdam.

,, NORMAN, CONOLLY, F.R.C.P.I., F.R.C.S.I., St. Dymphna's, N. Circular Road, Dublin.

1887 OGILVIE, GEORGE, M.B., M.R.C.P., 22, Welbeck Street, W.

,, OGILVIE, LESLIE, M.B., M.R.C.P., 46, Welbeck Street, W.

,, OLIVER, THOMAS, M.D., F.R.C.P., 7, Ellison Place, Newcastle-on-Tyne.

O.M. ORANGE, WM., C.B., M.D., F.R.C.P., The Bryn, Godalming, Surrey.

,, ORD, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C., 1893-94.)

,, ORMEROD, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-97.)

,, ORR, DAVID, M.B., County Asylum, Prestwich, Manchester.

,, PAGE, HERBERT WILLIAM, M.A., F.R.C.S., 146, Harley Street, W. (V.P., 1900-1; C., 1891-92.)

1895 PARKINSON, JOHN PORTER, M.D., M.R.C.P., 57, Wimpole Street, W.

1901 PARSONS, JOHN HERBERT, M.B., B.Sc., 28, Great Ormond Street, W.C.

1901 PASSMORE, E. S., M.D., M.R.C.P., The Asylum, Banstead.

1896 PATERSON, DONALD ROSE, M.D., M.R.C.P., 18, Windsor Place, Cardiff.

Elected

1895 PINDER, GEORGE, M.D., B.C., Hazeldene, Farncombe Road, Worthing.

1899 PIPER, FRANCIS P., M.B., M.R.C.S., L.R.C.P., Claybury Asylum, Woodford, Essex.

1888 PITT, G. N., M.D., F.R.C.P., 15, Portland Place, W.

O.M. POORE, G. VIVIAN, M.D., F.R.C.P., 24A, Portland Place, W. (C. 1898-99.)

,, PURVES, W. LAIDLAW, M.D., 20, Stratford Place, W.

1899 RAMBAUT, DANIEL F., M.D., B.Ch., Richmond District Asylum, Dublin.

1894 RANSOM, WILLIAM B., M.D., F.R.C.P., The Pavement, Nottingham.

1890 RAYNER, HENRY, M.D., M.R.C.P.Ed., 16, Queen Anne Street, W

O.M. REID, E. W., M.B., F.R.S., University College, Dundee.

1897 REISSMANN, CHARLES H., M.B., B.Sc., 7, Upper Montague Street, Russell Square, W.C.

1893 RENNIE, GEORGE E., M.D., M.R.C.P., College Street, Sydney.

1895 REYNOLDS, ERNEST S., M.D., F.R.C.P., 2, St. Peter's Square, Manchester.

1892 RICHARDS, JOSEPH PEEKE, M.R.C.S., 6, Freeland Road, Ealing, W.

1891 RIVERS, W. H. R., M.A., M.D., F.R.C.P., St. John's College, Cambridge. (C., 1897-98.)

1898 ROBERTSON, WILLIAM FORD, M.D., 7, Hill Square, Edinburgh.

1892 ROWE, EDMUND LEWIS, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

,, RUFFER, MARC ARMAND, M.D., Alexandria, Egypt.

1889 RUSSELL, J. S. RISIEN, M.D., F.R.C.P., 4, Queen Anne Street, W. (Sec., 1896-1900; C., 1901.)

1899 RUSSELL, J. W., M.D., M.R.C.P., 72, Newhall Street, Birmingham.

O.M. SANDERSON, Sir J. BURDON, Bart., M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.

,, SAVAGE, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (Pres., 1897; V.P., 1895; C., 1886-90, 1898.)

1888 SAVILL, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

O.M. SCHÄFER, E. A., F.R.S., University of Edinburgh. (Pres., 1893; V.P., 1892; C., 1886-98.)

,, SEMON, Sir FELIX, M.D., F.R.C.P., 39, Wimpole Street, W. (C., 1899-1901.)

1898 SEWARD, WM. JOSEPH, M.B., Colney Hatch Asylum, N.

O.M. SHARKEY, S. J., M.D., F.R.C.P., 22, Harley Street, W. (C., 1889-92.)

,, SHERRINGTON, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C., 1892-95.)

1894 SHUTTLEWORTH, GEORGE, E., M.D., Ancaster House, Richmond.

1898 SIMPSON, FRANCIS O., L.R.C.P., M.R.C.S., Rainhill Asylum, Liverpool.

1900 SINGER, H. D., M.D., National Hospital, Queen Square, W.C.

1895 SMITH, F. J., M.D., F.R.C.P., 138, Harley Street, W.

Elected

1887 SMITH, R. PERCY, M.D., F.R.C.P., 36, Queen Anne Street, W. (Editor of "Brain," 1901.)

1895 SPICER, W. T. HOLMES, M.D., F.R.C.S., 47, Welbeck Street, W.

,, STANSFIELD, THOS. E. K., M.B., London County Asylum, Bexley, Kent.

1895 STANLEY, DOUGLAS, M.D., 19, Easy Row, Birmingham.

1892 STARLING, ERNEST HENRY, M.D., F.R.C.P., F.R.S., 8, Park Square, N.W.

1897 STEWART, PURVES, M.A., M.D., M.R.C.P., 133, Harley Street, W.

,, STIRLING, WM., M.D., D.Sc., Owens College, Manchester.

1894 STODART-WALKER, A., M.B., F.R.C.P.Ed., 30, Walker St., Edinburgh.

1897 STODDART, WM. H., M.D., M.R.C.P., Bethlem Royal Hospital, Lambeth, S.E.

1895 STOUT, G. F., M.A., St. John's College, Cambridge.

1887 SUCKLING, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.

1889 SUTHERLAND, HENRY, M.D., M.R.C.P., 21, New Cavendish Street, Portland Place, W.

1888 SYERS, HENRY W., M.D., M.R.C.P., 75, Wimpole Street, W.

1898 TAYLOR, F. R. P., M.D., Darenth Asylums and Schools, Dartford, Kent.

1891 TAYLOR, JAMES, M.D., F.R.C.P., 49, Welbeck Street, W. (C. 1900-1.)

1895 TELFORD-SMITH, T., M.A., M.D., South Hill Park, Bromley, Kent.

1898 THOMSON, H. CAMPBELL, M.D., M.R.C.P., 34, Queen Anne Street, W.

1889 THORBURN, WM., F.R.C.S., The Limes, Nelson Street, Manchester. (C. 1901.)

1892 TITCHENER, E. B., B.A., Ph.D., 72, Heustis Street, Ithaca, N.Y.

O.M. TOOTH, H. H., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896-99.)

1892 TREVELYAN, E. F., M.D., B.Sc., M.R.C.P., 40, Park Square, Leeds.

1892 TUCKEY, CHARLES LLOYD, M.D., 88, Park Street, Grosvenor Square, W.

1892 TUKE, Sir J. BATTY, M.D., F.R.C.P.Ed., 20, Charlotte Sq., Edinburgh.

1891 TUKE, THOMAS SEYMOUR, M.B., Chiswick House, Chiswick.

,, TURNER, WILLIAM ALDREN, M.D., F.R.C.P., 13, Queen Anne Street, W.

1898 TURNEY, HORACE G., M.D., F.R.C.P., 68, Portland Place, W.

O.M. TWEEDY, JOHN, F.R.C.S., 100, Harley Street, W.

1888 VOORTHUS, J. A., M.D., Apeldoorn, 542, Loulaan, Netherlands.

O.M. WALLER, AUGUSTUS, M.D., F.R.S., 16, Grove End Road, N.W. (Pres., 1900; V.P., 1899; C. 1894-97, 1901.)

,, WARD, J., D.Sc., Trinity College, Cambridge.

,, WARNER, FRANCIS, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.

1892 WASHBOURN, J. W., M.D., F.R.C.P., 6, Cavendish Place, W.

Elected

O.M. WATTEVILLE, A. DE, M.A., M.B., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896-1900; Sec., 1886-89; Editor of *Brain*, 1880-1900.)

1897 WEBER, F. PARKES, M.D., F.R.C.P., 19, Harley Street, W.

,, WEST, SAMUEL, M.D., F.R.C.P., 15, Wimpole Street, W.

1897 WHITE, ERNEST W., M.B., M.R.C.P., City of London Asylum, Dartford.

O.M. WHITE, W. HALE, M.D., F.R.C.P., 65, Harley St., W. (C., 1897-1901.)

1894 WHITING, ARTHUR J., M.D., M.R.C.P., 142, Harley Street, W.

1889 WIGLESWORTH, JOSEPH, M.D., F.R.C.P., County Asylum, Rainhill, Lancashire.

1900 WILLIAMS, LEONARD, M.D., 8, York Street, Portman Square, W.

1894 WILLIAMSON, RICHARD T., M.D., F.R.C.P., 294, Oxford Road, Manchester.

1896 WILSON, ALBERT, M.D., M.R.C.P., Minto House, South Woodford, Essex.

,, WOOD, GUY M., M.B., M.R.C.P., 6, Woburn Square, W.C.

1889 WOOD, T. OUTTERSON, M.D., M.R.C.P., 40, Margaret Street, Cavendish Square, W.

1892 WOODHEAD, G. IMS, M.D., F.R.C.P.Ed., 6, Scrope Terrace, Cambridge.

1899 WRIGHT, HAMILTON, M.D., Pathological Institute, Selangor Straits Settlement.

1898 YOUNG, ROBERT ARTHUR, M.D., B.Sc., M.R.C.P., 41, Wimpole St., W.